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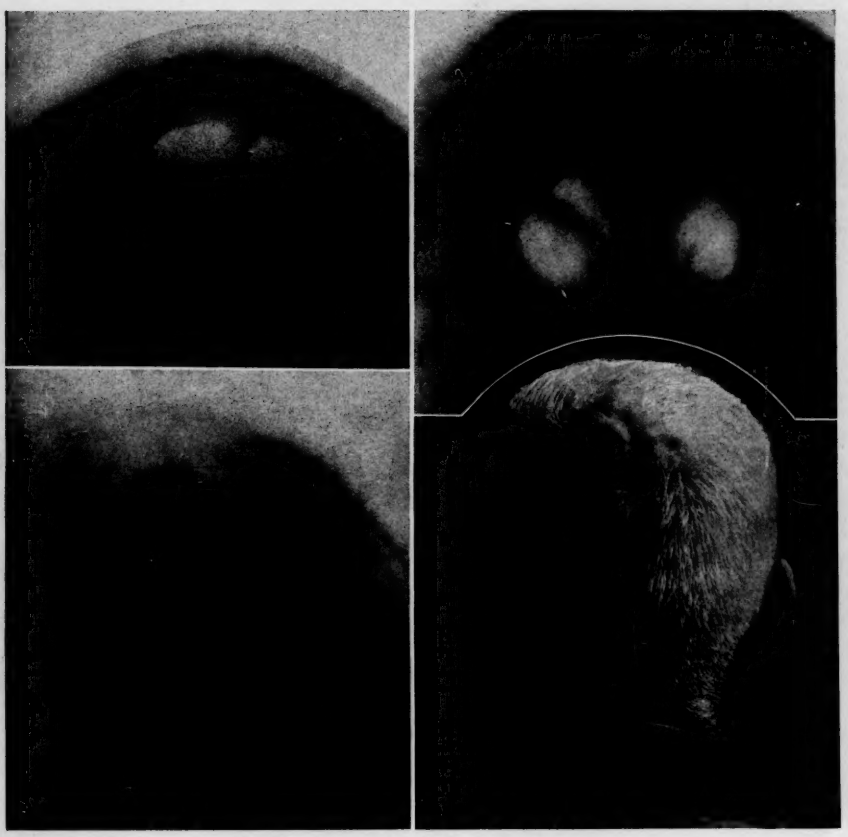

INHERITED "EYES" IN THE SKULL
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"Dwarf-Eye" in Chickens

Race and Hair-Form

A New Chromosome Map

How Size-Genes Function

Eugenics Section

VOLUME 32 SEPTEMBER, 1941 NUMBER 9

The American Genetic Association

An incorporated organization devoted to promoting a knowledge of the laws of heredity and their application to the improvement of plants, animals, and human racial stocks

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In cooperation with the American Eugenics Society

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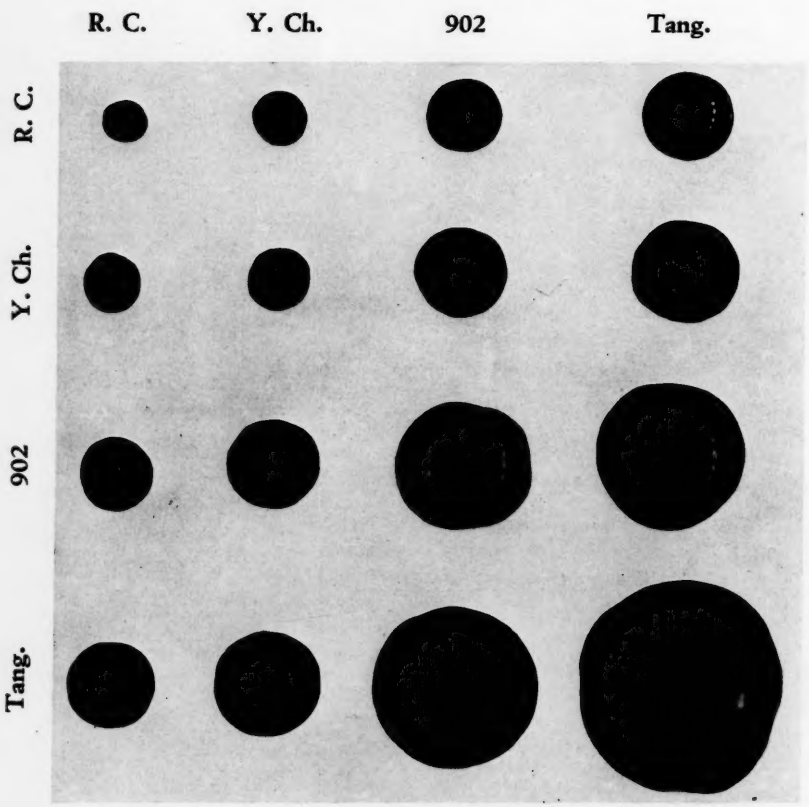

INTERACTION OF SIZE GENES

Frontispiece

Fruits of four varieties and their 12 F_1 hybrids by diallel crossing, as reared in nine-inch pots in the greenhouse. Reciprocal crosses are alike in size. The mean fruit weights were in general lower indoors especially for large-fruited types, than in the field (Figure 3), the averages of three replications being in order in successive rows left to right: 0.95, 1.6, 4.6, 6.5; 1.6, 2.1, 8.5, 13.4; 4.5, 9.1, 26.0, 40.0; 6.8, 12.7, 30.2 and 55.0 grams. The orderly progression in size changes observed in these hybrids demonstrates that the end-products of different size-gene interactions in this material are geometric in their expression, not merely additive.

SIZE INHERITANCE IN TOMATO FRUITS

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THE standard explanation of size inheritance by multiple factors, which are assumed to act without dominance and with equal and additive effects, may be fairly criticized as certainly inadequate and probably basically erroneous. This theory has doubtless served well as a first approximation to explain the majority of cases in a rough and superficial way that was useful in a practical sense, just as Newtonian principles long sufficed in Physics. But the examples most frequently encountered and analysed, such as length of corn ears or tobacco corolla tubes, or weights of beans or mice, are, after all, not really critical, because the parental size differences are too small to show up clearly the essential underlying processes. More appropriate materials for a rigorous analysis of size inheritance have been found and used, where the parents differ in size by four or ten or a hundred to one. Such wide ranges in size are available in fruits of tomato, pepper, or apple varieties and species, and in the body weights of breeds of dogs, horses, or domestic fowl.

Studies of these favorable materials, especially tomato fruit size, reveals what seems to be the more generalized form of inheritance of size differences, of which corn ear length, for instance, is only a particular and very unrepresentative example. In the newer findings marked size effects are often traced to known, typically dominant or recessive qualitative genes; these specific effects may be large or small; and, especially, the effects are proportional or geometric in nature, rather than additive. So far as the critical evidence goes, size genes singly or groups of size genes collectively almost certainly do not add so many grams to a fruit or body weight or so many centimeters to an ear length, but instead cause a certain percentage of increase in weight or length, the absolute amount of the increase depending closely upon the basic size already attained or determined by other factors.

Such a proportionate effect of size genes is demonstrable in various ways. It has already been described (1) for *single genes* controlling other visible qualitative characters (e.g. 'lutescent', *l*, and 'fasciated', *f*, in the tomato); (2) for the genes of a *chromosome* linked with a qualitative marker (e.g. the size genes of chromosome I of the tomato linked with 'dwarf', *d*); and (3) is demonstrated in this paper for the total size effects of a *genome* substitution. It is also indicated (4) in the approach of F_1 and F_2 size means to the geometric mean of the parents, and in the many cases where F_2 and back-cross distributions are made normal by logarithmic plotting. Moreover (5) it is consistent with this view that selection for larger size has proven far more efficient even in the house mouse than has selection for smaller size (unpublished). If size inheritance is really geometric in nature, then interpretations of selection experiments dealing with size or other similar quantitative characters will need radical revision as to the number of factors concerned, the frequency of mutation of these factors, and the expected efficacy of plus and minus selection.

A Latin Square Experiment Using Diallel Crosses

Plan of the Experiment

In 1939, seeds of four tomato varieties and the six F_1 crosses between them were planted simultaneously (1940) in the greenhouse at Vineland Horticultural Experiment Station, transplanted to flats and removed to the field into a 10 × 10 Latin Square with border plants. Data taken included the weights of 10-20 mature fruits per plant. The randomized arrangement of the square and the mean weight of fruits of each of the 100 plants are shown in Figure 1.

Each of the four varieties has been selfed for 12-20 generations and is obviously highly homozygous for all characters. All of the varieties bear approximately spherical (slightly oblate) 2- (rarely 3-) loculed fruit of similar struc-

1 1.0	10 110.0	6 8.4	2 4.0	7 11.8	9 21.4	4 104.4	5 2.0	3 52.0	8 15.8
2 3.8	9 20.6	5 2.0	10 100.0	8 16.2	1 0.6	6 8.4	3 51.0	7 12.6	4 100.0
3 65.0	1 0.6	4 99.0	7 10.6	6 8.8	10 96.4	8 16.0	9 19.4	5 1.8	2 4.0
4 101.0	2 3.6	3 53.0	1 0.8	5 1.4	6 8.0	7 9.4	8 16.8	9 19.6	10 87.5
5 1.5	8 13.6	1 0.9	4 98.0	9 17.4	7 8.4	10 87.0	6 6.0	2 3.6	3 49.6
6 8.0	3 47.0	2 4.0	5 1.0	10 111.0	8 16.2	9 18.4	7 9.2	4 144.0	1 0.6
7 15.0	4 104.0	8 14.4	6 6.8	3 50.0	5 1.8	2 3.4	10 98.0	1 1.0	9 20.0
8 8.6	5 1.8	7 9.0	9 18.2	4 92.0	3 56.8	1 0.6	2 3.6	10 98.8	6 8.2
9 19.4	6 8.2	10 106.0	3 58.0	1 1.0	2 2.8	5 1.4	4 113.0	8 11.5	7 11.0
10 103.7	7 10.0	9 19.8	8 15.0	2 3.4	4 113.3	3 66.6	1 0.8	6 7.0	5 2.2

FRUIT-WEIGHTS OF TOMATO VARIETIES AND HYBRIDS

Figure 1

The 10 × 10 Latin Square, containing one hundred plants of four tomato varieties and their six F_1 hybrids: (1)=R.C. (Red Currant); (2)=Y.Ch. (Yellow Cherry); (3)=902 (*dry calh* selection); (4=Tang. (Tangerine, *tuy*); and (5)=R.C. × Y. Ch.; (6)=R.C. × 902; (7)=R.C. × Tang.; (8)=Y.Ch. × 902; (9)=Y. Ch. × Tang.; (10)=902 × Tang. Numbers below each square show the mean fruit weight of the plant.

ture. Red Currant has wild-type factors and 0.8 gram fruits; Yellow Cherry has yellow, 3.6 gm fruits; 902 carries recessive markers for chromosomes I to VII, including yellowish (lutescent) foliage, and has nearly white, 54.9 gm fruit; Tangerine carries three recessives and its tangerine colored fruits averaged 106.8 grams. The only objectionable factors, noticeably affecting fruit size, are the lutescent foliage gene in 902, which hinders both plant and fruit growth and reduces average fruit weight by about 20%, and the rather frequent extra locule in 902 and Tangerine and their hybrids, which increases the fruit size by a small amount.

The twelve possible hybrids between the four varieties have been made and compared both in the greenhouse and in

		Pollen Parent			
		R.C.	Y.Ch.	902	Tang.
Seed Parent	R.C.	*R.C.* / R.C. / 0.8	*Y.Ch.* / R.C. / 1.7	*902* / R.C. / 7.8	*Tang.* / R.C. / 10.7
	Y.Ch.	*R.C.* / Y.Ch. / 1.7	*Y.Ch.* / Y.Ch. / 3.6	*902* / Y.Ch. / 14.4	*Tang.* / Y.Ch. / 19.4
	902	*R.C.* / 902 / 7.8	*Y.Ch.* / 902 / 14.4	*902* / 902 / 54.9 (65.9)	*Tang.* / 902 / 99.8
	Tang.	*R.C.* / Tang. / 10.7	*Y.Ch.* / Tang. / 19.4	*902* / Tang. / 99.8	*Tang.* / Tang. / 106.8

EFFECT OF GENOME SUBSTITUTIONS ON TOMATO FRUIT WEIGHT (GRAMS)

Figure 2

The genetic constitution of four parents and their hybrids, and their observed mean fruit weights from the Latin Square. The 902 weight is corrected (figure in parenthesis) by adding 20% for the effect of lutescence. This correction is not essential to the interpretation offered; if 902 and its hybrids are omitted, the results are further regularized.

the field. The Frontispiece shows photographs of fruits from plants raised indoors in nine-inch pots in 1940.

Reciprocal crosses have been repeatedly shown to be alike in fruit size, there being no detectable maternal or cytoplasmic influence. Accordingly only six hybrids were used in the Latin Square. This kept the square within the proper size for the purpose.

Results

The mean fruit weights of the parent varieties and hybrids in the Latin Square may be summarized as in Figure 2.

Analysis of the variance in fruit weights shown by the plants of the Latin Square (Figure 1) permits separation of the variance due to environment (soil, etc.) as about 0.4%, that due to heredity (varieties) as 99.4%, and the error portion as 0.2%. Fruit size is thus controlled almost exclusively by genotypes under the relatively uniform conditions obtaining in the Latin Square.

The most important results for the present purpose appear from further study of Figure 2. The fruit weights of the parent and hybrid plants are not chaotic and irregular, but reveal distinctly orderly and predictable relations. (1) As before reported the F_1 fruit weights are not intermediate in size but closely approach the geometric means of the parental varieties; e.g. F_1 plants from R.C. $\times$ Y.Ch. bear fruits averaging the square root of 0.8 $\times$ 3.6, or 1.7 grams.

	F_1 fruit weights in grams		
F_1 hybrids	observed	geom. mean	arith. mean
R. C. $\times$ Y. Ch.	1.7	1.7	2.2
R. C. $\times$ 902	7.8	7.3	33.3
R. C. $\times$ Tang.	10.7	9.2	53.8
Y. Ch. $\times$ 902	14.4	15.4	34.7
Y. Ch. $\times$ Tang.	19.4	19.6	55.2
902 $\times$ Tang.	99.8	83.9	86.3

Only in the F_1 from 902 $\times$ Tang. does the fruit weight depart greatly from the geometric mean (calculated with the corrected figure for 902). Hybrids between large parent varieties not very unequal in size often show all the irregularities reported in such cases in the

older literature; in these cases the control of fruit size probably passes to minor and accessory genes and interactions.

(2) The chief result we wish to stress is found by systematic comparison of the fruit weight data in Figure 2. Compare columns one and two, and note that at each level the genotype in column one is changed in column two merely by the substitution of a yellow cherry genome (here the single set of chromosomes contributed by one gamete) with its size genes for a corresponding genome from red currant. In all the four comparisons this substitution regularly causes the fruit weight to be about doubled whatever the residual genotype may be; but in doubling the weights the absolute weights are raised by amounts ranging from only 0.9 gram in the first case to 1.9, 6.6 and 8.7 grams in the other combinations. A given set of size genes thus produces the same phenotypic effect in each of the four instances, if that effect is defined and measured in geometric terms, but not if the effect is described in the arithmetic terms ordinarily employed.

Similar comparisons of the second and third columns show that substitution of a 902 for a yellow cherry genome raises the fruit weight in parents or hybrids by about five times, though the absolute increase is as low as 6.1 grams in the first and as high as 80.4 grams in the last case. The increases from column one to column three average 9.1 times, from columns one to four, 11.3 times, from three to four 1.2 times.

Discussion

Tomato fruit size depends upon the growth and differentiation of the meristematic tissue of the fruit anlage, which enlarges by a series of cell divisions (largely before anthesis) and by cell expansion (mainly after anthesis when the pollen auxin effect is expressed). The limits of growth are set by both the surrounding environmental conditions and by differences in hereditary factors. By reducing the environmental inequalities to a minimum, and by avoiding unnecessary and complicating shape factors, which practically defy a satisfactory analysis (Powers '39), we have attempted to discover the combined phenotypic effect and average mode of action of a whole set of size genes.

The somewhat novel method consists of making up the series of diallel crosses between carefully chosen, long-inbred parent varieties, and then arranging and systematically comparing the fruit weights of the parents and F_1 hybrids. The full series proves to be distinctly orderly and unexpectedly simple in interpretation. The findings reported would appear to demonstrate once and for all that the total differential effect of replacing one set of size allels for another is geometric and not additive in nature. The same genome substitution regularly produces the same phenotypic effect upon fruit size, when that effect is described in geometric terms on a strictly proportional basis, as a percentage increase or decrease. These results accordingly confirm and extend those previously reported by other methods.

In such a situation small size might be considered a more or less dominant character, as has been proposed in a number of instances. This view is purely descriptive, however, and by itself explains nothing, though it is consistent with and indeed requires a developmental or physiological explanation such as has already been provided by histological and genetic work.

A further point in favor of the geometric interpretation of size phenomena has been too much neglected. The linear, surface and volume measurements, even of one and the same perfectly spherical fruit, cannot *all* be arithmetically intermediate between the parents, since they depend respectively upon the first, second and third powers of the linear diameter. Yet, like Procrustes' guests, all such measurements have usually been forced to fit an arithmetic interpretation, which is impossible in actual application as it is unlikely in theory. A geometric explanation consistently avoids this paradox. In biological affairs size depends on cell numbers and cell sizes; cell numbers increase geometrically by mitosis

from 1 to 2, 4, 8, etc., the rate of progression being 2, and cell expansion has been shown also to be controlled geometrically (see Butler[1] for an analysis of F_2 segregation in cell sizes). Since it seems probable that a great many physiological processes are themselves more or less directly dependent on cell number and/or cell size, they, as quantitative characters, would also be expected to rest on the same basis as does size.

The chief observations described in the main experiment might be restated and tentatively explained in more physiological terms. When one genome is substituted for another, the one from the larger sized parent may supply to the seed through the size genes of its chromosomes, additional or more active substances (e.g. enzymes) which control the rate or number of cell divisions and the amount of cell expansion during fruit growth the next generation. The effect of such enzymes would be proportional to the amount of substrate (number of cells of a certain size) already present or determined by other factors. The resulting extra growth activity induced by the genome substitution would then be proportional to the amount of substrate upon which the activating substances act. In such a system the amount of substrate becomes a factor limiting size.

Conclusion

Evidence from a series of diallel crosses raised in Latin Square confirms the view that size genes determining fruit weight in the tomato act in a proportionate or geometric way.

Literature Cited

The chief relevant papers are referred to in:

1. Butler, L. The Inheritance of Fruit Size in the Tomato. *Can. Jl. Research, C.* 19: 216-224. 1941.

2. MacArthur, J. W., and L. Butler. Size Inheritance and Geometric Growth Processes in the Tomato Fruit, *Genetics* 23:253-268. 1938.

✦

A NEW FUR PATTERN VARIATION IN RATS

King* has reported the development of hooded patterns and curly haired fur, both of which were mutations, in Norwegian gray rats.

The strain of animals used in this laboratory is mixed with regard to fur color and pattern. Albino, black, black hood, buff and buff hood are the coat color variations which have followed the Mendelian Laws during the time that records have been kept on some 20,000 individual progeny comprising almost 3,000 matings.

One black male animal from the fifth litter of a black hooded female, mated to a male with buff colored fur, exhibited a unilateral white area on the antero-dorsal portion of the head, extending medially from the nose to the eye (see Figure 3). All other litter mates had normal fur patterns.

FACE BLAZE
Figure 3

This type of variation in rats has been hitherto unreported and is the first of any kind observed in this laboratory. Attempts are now in progress to reproduce this condition to determine whether or not it is a true mutation. The results of these experiments will be included in further reports.

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Kenneth L. Cartwright
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*King, Helen D. *Life Processes in Gray Norway Rats During 14 Years in Captivity.* American Anatomical Memoirs No. 17.

A TRISOMIC GRASSHOPPER

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WHEN the chromosome complement of an organism is not an exact multiple of the haploid set, the organism is termed aneuploid. This condition has been found quite frequently in plants, but among animals, apart from the unusual and specialized sex chromosomes of some of the Hemiptera[5] and certain cases of inert chromosomes, *Drosophila melanogaster* is the only species in which the same state of affairs has been observed.[8] Probably the main reason for this anomaly is that the sexual reproduction of triploids, which is the source of most aneuploids in plants, is rare in animals.

Among thirty males of the acridioid Grasshopper *Mecostethus grossus* Linn. collected in the New Forest, England, in 1940, one individual was found in which, in all mitoses examined, the third largest autosome was represented three times instead of twice. Normally the diploid complement of the male *Mecostethus* consists of 22 nearly telocentric autosomes and a single sex chromosome. This individual possessed 23 autosomes and a sex chromosome and was therefore a trisomic aneuploid.

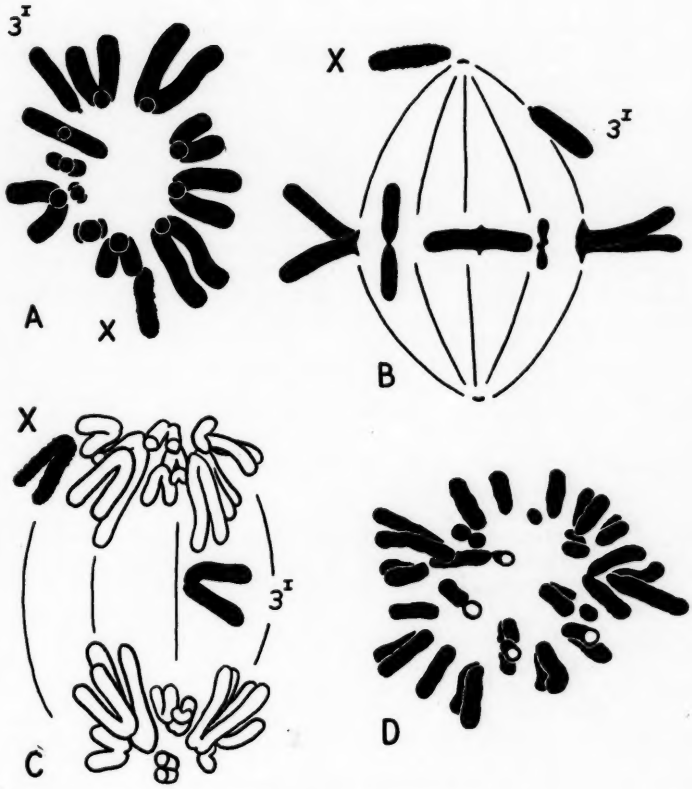
Mecostethus grossus is an organism showing a very special distribution of crossing-over as seen at first meiotic metaphase. This was illustrated by McClung[10] (cf. White[11]). In many individuals each bivalent is held together by only one chiasma, which is placed very close to the centromere. A consequence of this proximal localisation is that in spite of the fact that in the trisomic there are three homologous 3rd chromosomes, these never associate to form a bivalent. They are always present as bivalent and univalent; clearly it is a matter of chance which one of the three, and they are cytologically indistinguishable, is left as univalent (Figure 4*A*).

The univalent third autosome is readily distinguishable from the heterochromatic *X*-chromosome at first meiotic metaphase. It shows more clearly than the bivalents that the centromere is not quite terminally placed (Figure 4*A* and 4*B*). Darlington[4] has pointed out that in the grasshoppers *Chortippus* and *Stauroderus* what appear to be single-armed chromosomes in reality are not telocentric since they form chiasmata in their short arms.

Now in trisomic *Mecostethus* we have two kinds of univalent. The behavior of the univalent autosome is therefore to be contrasted with that of the univalent sex chromosome at first meiotic metaphase. The centromere of the sex chromosome appears to be almost inactive at this division; the sex chromosome does not congregate with the bivalents at the equator, but lies passively at one pole and seems never to attach itself to the main spindle.

The univalent autosome lags behind the bivalents in congression on the metaphase plate. It may not get on to the plate at all, in which case it behaves like the sex chromosome and is passively included in one telophase group (Figure 4*B*). If the univalent autosome succeeds in reaching the plate, it remains there after the anaphase movement of the autosome bivalents has begun (Figure 4*C*). Lapse of chromatid attraction in the univalent autosome coincides with the onset of this condition in the bivalents. This is not always true of the *X*. The chromatids are carried far apart as the spindle stretches, but they remain attached to one another at the undivided centromere. Unlike univalents in Kihara's[7] *Triticum-Aegilops* hybrids or the sex univalent in *Odonata*[1] the centromere of the *Mecostethus* univalent never divides at the first meiotic division.

When the autosomal univalent is left at the equator the anaphase spindle fails to elongate in normal fashion. Whenever the univalent has become associated with the spindle, the telophase groups are apparently not free to move apart, with the result that during despiralisation and nuclear membrane formation a diploid restitution nucleus is formed. Thus the univalent can never be lost.



MEIOSIS IN TRIPLOID

Figure 4

A-C. 1st meiosis. *A*—Polar view of metaphase in trisomic *Mecostethus,* (X=sex univalent, 3^{I} = autosomal univalent). *B*—Side view of metaphase. Autosomal univalent not on the plate. *C*—Side view of anaphase with univalent lagging at the equator. *D*—Polar view of diploid second meiotic metaphase with 23 autosomes + *X*. (×2000.)

This is in complete contrast to the effect of undivided univalents in *Triticum* reported by Matsumoto,[9] where the spindle undergoes super-elongation.

As a consequence of this behavior 50 per cent of the second meiotic metaphases are found to be diploid, with 24 chromosomes (Figure 4*D*). This is an estimate of the frequency with which the autosomal univalent reaches the equatorial plate at the first meiotic division. The haploid divisions contain 11, 12 or 13 chromosomes according to the independent segregation of the sex and autosomal univalents. Second anaphase proceeds regularly in all types of second spermatocyte, and leads to the formation of second spermatids and sperm of the following constitutions:

Complement	*Proportion*
23*A* + *X*	4
12*A* + *X*	1
11*A* + *X*	1
12*A*	1
11*A*	1

It is possible to recognize spermatids of two sizes, the larger kind being diploid. There is no evidence of degeneration of spermatids or sperm.

The trisomic *Mecostethus* was externally a normal male, with nothing to suggest a subnormal viability. This affords an interesting comparison with *Drosophila melanogaster,* where Li[8] found trisomy of the large autosomes was always lethal at an early stage, whereas trisomy of the minute fourth autosome led to slight reduction in viability. It must be remembered that *Mecostethus* possesses many more chromosomes than does *Drosophila melanogaster.* In fact the third autosome, though large, constitutes only 1/16th of the chromatin bulk at meiosis.

The origin of the trisomic can be almost certainly ascribed to "non-disjunction" occurring in its male or female parent. White[11] has described failure of pairing at meiosis in a normal male *Mecostethus* and this can readily lead to non-disjunction.

Assuming that the unbalanced sperm of this trisomic were fully functional, and we have no evidence in animals that selection occurs during the male gamete stage, it could have given rise to a proportion of trisomic progeny whose survival would depend on their relative viability during development.

Diploid 2nd spermatocytes and sperm have frequently been reported in insect testes (Darlington,[4] Callan,[3] et alii). They can be produced in a variety of ways. They would give rise to triploids. Triploid individuals in a normally diploid animal species, however, have been rarely found. (*Triturus viridescens*,[6] *Triton taeniatus*,[2] and *Triton vulgaris* [Barber and Callan, unpublished] are known examples). Apart from unbalancing the sex determining mechanism in some organisms, the main reason for this appears to be that since far more male than female animals are subject to cytological investigation, triploids would only be found in organisms with male homogametv. Though interesting as an individual, it is therefore unlikely that this *Mecostethus* trisomic would have provided suitable material for any evolutionary step.

Summary

A male *Mecostethus grossus* has been found which was trisomic for the third largest autosome. Owing to chiasma localisation in this species, trivalents do not occur. In 50 per cent of the 1st spermatocytes the univalent third chromosome behaves like the *X,* and is included undivided in one telophase group. In the remaining 50 per cent the univalent congresses on the metaphase plate later than the bivalents. It never divides, and by interfering with the elongation of the spindle causes diploid restitution second spermatocyte nuclei to be formed.

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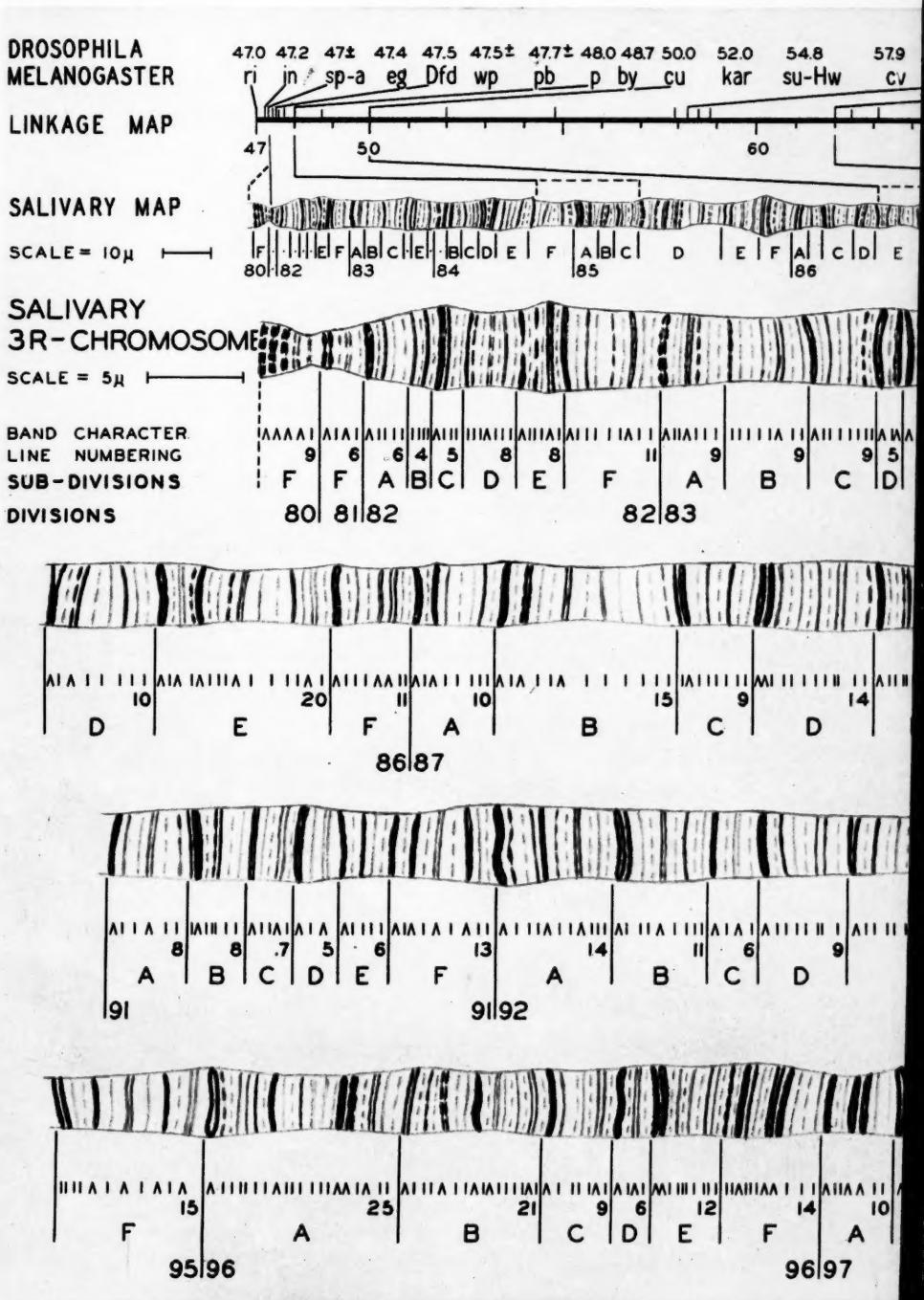

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57.9 cv
58.2 Sb
58.5 ss
58.8 bx
62.0 sr
62.4 Mf
63.1 gl
64.5 sed
66.2 Dl
69.5 H
70.7 e
75.7 cd
76.2 wo
79.7 Mw
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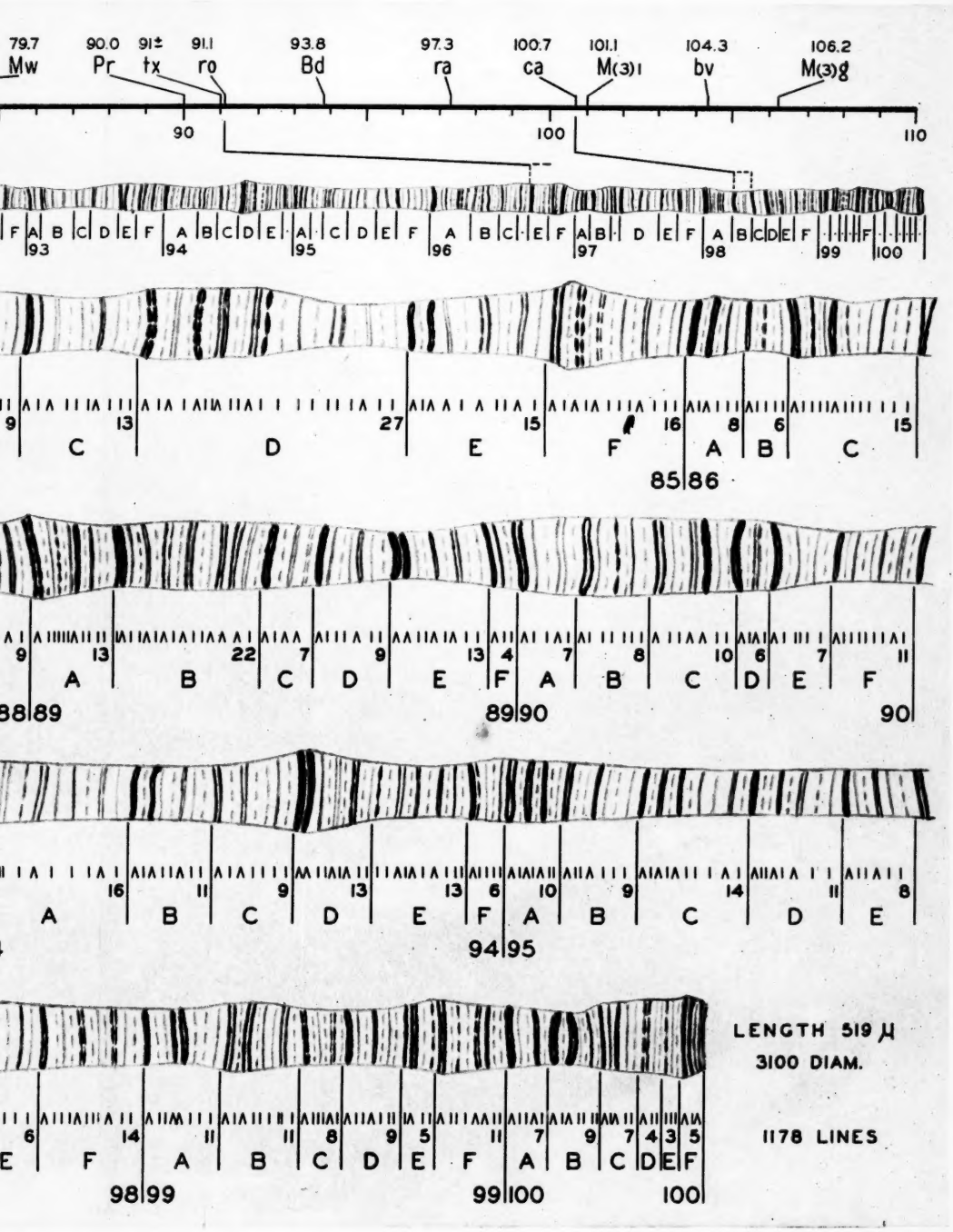

MAP OF THE SALIVARY GLAND 3R CHROMOSOME OF *DROSOPHILA MELANOGASTER*.

Figure 5

A REVISION OF THE SALIVARY GLAND 3R-CHROMOSOME MAP

Of *Drosophila Melanogaster*

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THE present map is the fourth in a series, made possible by a grant from the Carnegie Institution of Washington, of revisions of the salivary gland chromosome maps of *Drosophila melanogaster*. Preceding this map of the 3R-chromosome, revisions were published of the *X*- (this JOURNAL, 1938),[2] of 2R (1939),[5] and of 3L (1941).[6] All of these revisions serve as supplements to the original map published in 1935,[1] each one retaining the divisions and subdivisions substantially as in that map, with necessary changes clearly indicated.

Permanent aceto-carmine smears of larval salivary glands, prepared by Bridges' vapor replacement method as described in *Stain Technology*,[3] was the material used for this revision, as in the former ones. Chromosomes were selected for favorable conditions of stretching and staining, and an average of ten camera lucida drawings for each section was made from these preparations at a magnification of 5,000 diameters. The revision consists of a generalized composite of these drawings, its dimensions being averages of their lengths and widths, with the individual bands being filled in by comparison of all the drawings. Wherever necessary, reference was made to the original preparations and to others selected for checking purposes, the actual structure found to be the most closely compatible with these averages being taken as the standard for the map.

Many more bands than those indicated in the 1935 map have been included in the revision, and the characterization of the bands has been improved; many originally single bands have now been found to be doublets or capsules, many capsules have been resolved into close doubles, and many faint bands have been added. The present map, being an average, represents a chromosome of medium stretch, hence allowance for differences in proportion and amount of stretch must be made, in that more lax preparations will not show as many bands, nor as many resolved doubles, and more extended ones may change the general aspect of intensity and relationship. In general, however, the present map is adequate for all special studies, without the need of a revision being made for each new problem.

The present limits of the major divisions, as marked on the 1935 map, have been retained in every case. One or two subdivision lines have been moved a band or so, to make use of darker ones, but the changes are so slight as not to be indicated on the revision. Several preparations were found which were complete through the chromocenter, and it was possible to map the whole of that region. Section 80*F* has been redrawn from the earlier 3*L* chromosome revision,[6] but its bands have not been included in the band count. The 3R revision, then, starts with section 81, which contains but one subdivision (in order to preserve the seriation of the 1935 map), all the chromocenter being included in section 80 of 3*L*. As indicated in that earlier revision, the spindle-fiber attachment is between 80*E*1.2 and 81*F*1.2.

The new map contains 1178 bands, counting capsules as two, compared to the 725 of the 1935 map, and is 519 microns long as against the 275 microns of the old. Choice of larger chromosomes, and use of sections of greater and more uniform stretch account for the differences in these dimensions.

The most constant features of the 3R-chromosome are the rather bulbous "cucumber" base, the puff of 85*F*, the dif-

*Fellow, Carnegie Institution of Washington.

fuse double bands 88*A*4.5 and 9.10, which make 88.*A* a landmark, the "duck's head" of 89*E*, the repeat in 90*D*, the series of heavy bands of 93*E* and *F*, the bulge of 94*D*, the general slim tapering of section 95, the heavy bands of section 98, which tend to bunch and disarrange the chromosome, without synapsing with each other, however; the heavy doublets of 100*B*, and the diffuse dark bands at the "goblet" tip.

The linkage map, first line of the figure, gives the latest revision of gene positions, as given in *DIS*-9 and the later corrections, only RK. 1's and 2's being included. Below that is the present salivary chromosome revision reproduced at one-fourth the size of the detailed map. Between the two as many correlations as possible are shown of the salivary gland chromosome positions of the genes, these positions being located according to data given in the symbol list of *DIS*-9, and in the symbol list revisions of *DIS*-12, making use of the information on inversion, translocation, and deficiency limits and inclusions.

The large detailed salivary gland map revision of the 3*R*-chromosome of *Drosophila melanogaster* follows on the last four lines of the figure.

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✦

Genes and Regeneration

Returning to the main question, it is clear, as was long ago argued, that a large proportion of plant cells manifest, through the products of their division, the possession of a genotype equivalent to that of the race. It is far from being demonstrated, however, that all cells of all plants are in this sense totipotent. The opposite possibility is suggested by the fact that, so far as experiment has shown, the cells of some tissues can give rise only to certain parts of the plant. Conspicuous illustrations are the failure, in the majority of angiosperms tested, of leaves, roots, and root meristems to regenerate complete plants. On the other hand, to be sure, the development of shoots from root cuttings is regular practice in the cultivation of a fair number of species. Doubtless the widespread power of proliferation may by improved experimental methods be shown to be more nearly or quite universal. But for the present it must be admitted that genotypic comprehensiveness seems to vary as between the cells of different tissues, of different organs, and even more between cells of corresponding tissues in different species.

This conclusion, if it becomes a conclusion, contradicts the expectation derived from the general uniformity of nuclear structure throughout the life of a plant. There remains, as previously noted, the possibility of the occurrence of genetically effective cytoplasmic changes during ontogeny. But in our present state of ignorance, a discussion of such conceivable changes would be futile.—Charles E. Allen, University of Chicago 50th Anniversary Symposium.

BILATERAL FENESTRAE IN THE PARIETAL BONES

More Cases of the "Catlin Mark"

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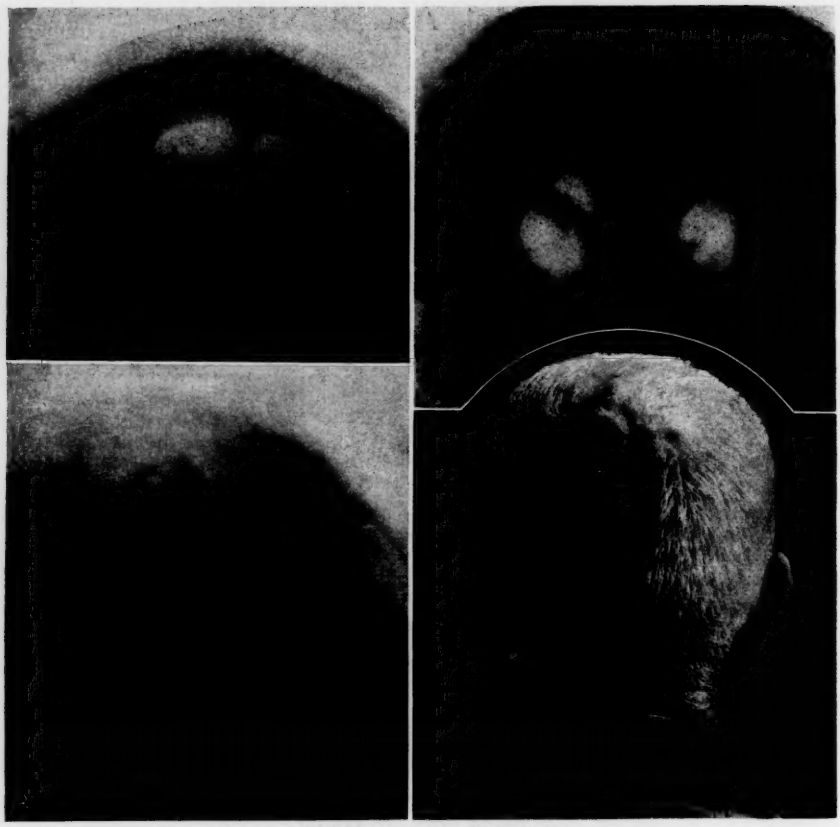
THE writer's interest in this strange anomaly began in 1922 when two of his students were observed to be "marked" with astonishingly large "windows" in their skulls (Figure 6). At that time this heredo-familial condition was considered very unusual, but it now appears that papers dealing with certain divisions of the subject-matter herein presented have been published as early as 1895[21] and the condition has received varied titles and designations, i.e.: the "Catlin Mark," "Large Bilateral Parietal Foramina," "Foramina Parietalia Permagna," or "Fenestrae Parietales Symmetricae," etc. Pepper and Pendergrass[19] who brought together some of this widely distributed material in 1936, summarize the writer's early paper as follows: "The most important report is that of Goldsmith in 1922. This escaped general medical notice because it appeared in the JOURNAL OF HEREDITY and also because the report had as its main title, 'The Catlin Mark' from the name of the family exhibiting the defect. It is true that the secondary title was 'The Inheritance of an unusual opening in the parietal bones.' Goldsmith had the opportunity to examine fifty-six members of the Catlin family distributed in five generations; sixteen exhibited the anomaly. Of these sixteen, three were women and thirteen were males."

The accompanying photograph and X-rays (Figure 6) illustrate the characteristic location and nature of the "Catlin Mark" as first reported by the writer. The openings, or fenestrae, are oval to round in adults, and have definite "healed" edges. In striking contrast, those of childhood are sometimes, if not usually, united making a large transversely elongated opening in the back of the skull (Figure 6, *A* and *B*). As ossification continues in the median region near the sagittal suture, the elongated opening in either parietal bone assumes its characteristic condition of the adult. (See Figures 6*C*, 7 to 9, and 12.) Zarfl's observations[24] confirm those of the writer. He followed the course of development of this anomaly in a baby from month to month. At first, there was a transverse elongated median defect. "By the growth of a median partition in the course of two years this picture changed and the two typical openings arose." Zarfl also described an infant skull from a Viennese collection which showed the same elongated single fenestra across the back of the head. This child might have shown the symmetrical fenestral condition had it lived to adulthood. Jonker[11] had an opportunity to follow the progress of ossification in an infant from eleven months to two years. When first observed, there was already a partial division into two openings, "but on the medial side they still remained in direct connection with the remainder of lozenge-shaped accessory fontanelle." Within one year after the first observation this accessory fontanelle had closed, leaving the typical fenestrae.

It is an interesting coincidence that in the same year (1922) the writer reported inheritance of the "Catlin Mark" in America, Neurath[16] reported a similar condition in Europe. The European worker described two cases, one a mother and her only child were affected, and in another family, a mother and two of her three children. No doubt Neurath would have found the defect in other members of these families if he had traced the family histories as did the author in his study of the Catlins.

Up to the present time, the European and American literature does not seem to have been brought together. As Halbertsma[9] says, "[prior to Goldsmith's paper] only one such hereditary case had been published, that of Symner describ-



"EYES" IN THE SKULL

Figure 6

Four views of the "Catlin Mark" as described by Goldsmith in 1922. *A* and *B* show X-rays of the "eyes" in the skulls of two grandchildren of the 70-year-old man whose head is shown in *C* and *D*. Note that the holes are so large that the scalp sinks in to form a noticeable depression (*D*). The child whose skull is shown in *B* is younger than *A*, as is indicated by the ossification in the median region to form the characteristic adult condition of the fenestrae, — two separate "eyes" instead of one large "window". With some "marked" children, this adult condition is present at birth, but in others a bridge appears in the midline during growth.

ing a father and son." Since no other members of this family were studied, the familial extent of the condition is unknown. Halbertsma shows that since 1930 only three papers describing the condition have appeared (Lelong and Bospuet,[12] Haszler,[10] and Wissler[23]). Since all these cases are European, and furthermore, since Halbertsma does not list a single paper from America, it is obvious that the European writers have been as negligent as the Americans in investigating the literature outside of their continent.

Referring only to European investigations (since he did not scrutinize the American journals), Halbertsma says, "The best observation, however, is that

AN ANCIENT "CATLIN MARK"

Figure 7

An ancient skull in the American Museum of Natural History showing "Bilateral Fenestrae in the Parietal Bones." This type of parietal foramina appearing in ancient skulls is classified as trephining. In a paper published elsewhere, the writer shows that such skulls are not illustrations of trepanation as is usually considered, but rather are inherited anomalies of the parietal bones in man.

NOT PREHISTORIC SURGERY

Figure 8

A skull with two holes in the parietal region, typical of many specimens mislabeled "trephining" in museums and private collections. It is a clear cut case of the "Catlin Mark." (From "Exploring Biology," Harcourt, Brace and Co. Specimen in U. S. National Museum.)

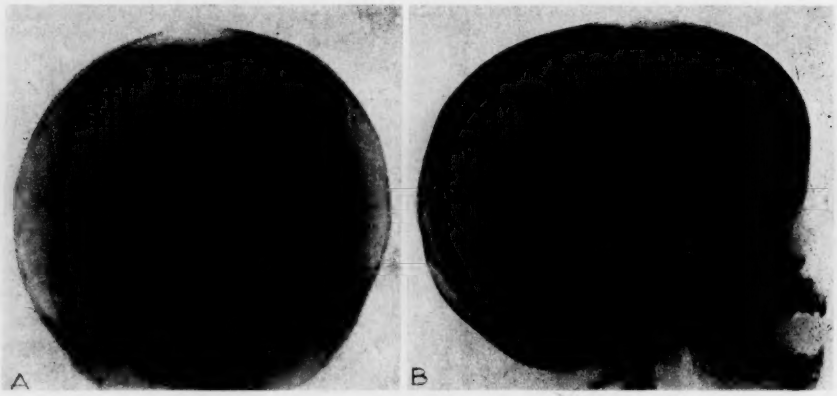
of Voigt (1937), who was able to study a family of fifteen persons in which the abnormality occurred in three generations." Marquezy[15] also recently described "lacunes congenitales de la voute cranienne" in an infant, the mother, and the maternal grandmother. The present description of this Dutch family is closely comparable to that of Voigt. "What distinguishes this family from most foreign observations is the absence of other congenital abnormalities."

This last remark relative to associated abnormalities also aptly applies to the high-grade Catlin family,[4] as the "mark" does not seem to be associated with any other human defect. In like manner, Halbertsma fails to find any other congenital abnormalities in the family he describes. Five out of a family of seventeen observed individuals showed "fenestrae parietales symmetricae" or the "Catlin Mark" of Goldsmith.

At the time of the publication of the "Catlin Mark" paper, scores of investigators were publishing observations on human inheritance so that "The inheritance of an unusual opening in the parietal bones" was merely another case of human inheritance meriting no unusual comment. It was considered merely as an isolated case. Recently (1936-1941), however, medical attention has been turned to this anomaly and numerous cases have been reported. Also a further study of the literature and of collections reveals a few references to isolated human skulls with "eyes in the back of the head."

One of the earliest references to the anomaly was by Greig as far back as 1892. Pamperl, in a paper presenting a striking case which he spoke of as "foramina parietalia permagna," claims that it had been observed only five times, in living persons or postmortem.

As was stated earlier, this defect probably attracted but little attention partly because it caused few if any symptoms except in an occasional case where pressure was applied or the patient received a stroke in that region. A backward fall by a student possessing the defect was the cause of the writer's discovering the



MONGOLISM AND "EYES" ASSOCIATED

Figure 9

A—X-ray of the head of an infant mongoloid imbecile showing a characteristic "Catlin Mark". Note that in this child the fenestrae were definitely formed at birth. *B*—Side view of same infant, showing position of the holes in the parietal bones and with reference to coronal, sagittal, and lambdoid sutures.

1922 case. The anomaly seems to be of no clinical importance except for a slight possibility that it might lead to a mistaken diagnosis in case of traumatic defects.

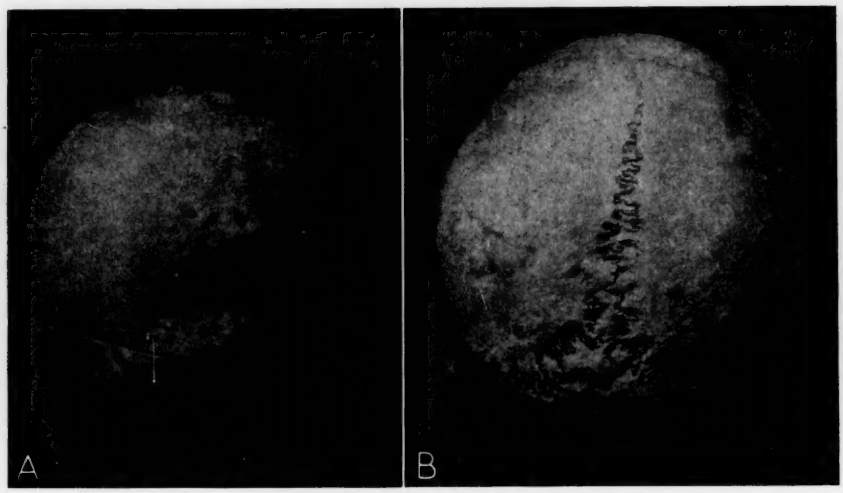
Ancient Skulls with Large Parietal Openings

In certain anatomical museums, skulls with large parietal openings have been observed for more than a century and a half. Greig refers to descriptions as far back as that of Lancisi in 1707. He mentions two such skulls in the Museum of the Royal College of Surgeons of Edinburgh. Voigt[22] refers to Lobstein's description of such anomalous skulls published in 1772. Hrdlicka and others have in their collections skulls from Indian burial mounds which show these large holes. Figure 7 is a pen sketch of a seemingly very old skull showing this defect. This is on public exhibition in the American Museum of Natural History in New York City. Another splendid specimen from the National Museum (Figure 8) has found its way into high school text books. A survey of various museums and private collections of human skulls shows that this so-called "Catlin Mark" type of anomaly is not uncommon. These symmetrically paired openings of the parietal bones are ordinarily classified as "trephining" in Museum collections. In the light of present knowledge such cases, which exactly duplicate the situation in living subjects showing the "Catlin Mark" must be reclassified, as such cases obviously give no proof of "healing" of the skull following the ministrations of ancient surgeons.

Another Living Case — A Mongoloid with the "Catlin Mark"

While the tendency is to unearth cases of the anomalies showing heredity tendencies, I wish to record an interesting case from Indiana which seems to be strictly non-familial. The X-ray plates shown in Figure 9 are of a child ten and one-half months old. The parents are of superior American stock, the father a man of distinction in the medical profession. Both parents have had X-rays made of their skulls, but find nothing unusual. Neither can the abnormality be found in any relative.

Another important feature of the case is that the boy is a mongoloid. Greig and others have cited cases wherein the



VARIATION IN VEIN DUCTS

Figure 10

A—"Normal" skull showing the two parietal foramina through which the emissary veins pass. *B*—A skull with only one foramen in center. In other skulls the foramina are lacking. The "Catlin Mark" appears not to be enlarged foramina. (University of Dubuque museum.)

so-called "enlarged foramina" are associated with other defects, — mental, mongolism, etc. Since this boy is from a superior family (as is the case with many other families showing individuals with the anomaly) and still is mongoloid, the case further contributes to the conclusion that the strange anomaly may or may not be associated with other human defects. The father of this "marked" mongoloid boy writes, "I have come to wonder whether mongolism and the Catlin mark may have more in common genetically than might at first be apparent. Might not the genetic defect which bobs up here and there in an unpredictable way to produce the 'mark' also carry the markings of a mongol? Might not the mongolism aspect of it be one of these tendencies, like the cancer-tendency in mice, which shows itself with advancing age and only rarely in a young subject?"

The father also mentions another possibility which may or may not be concerned with the anomaly under consideration: "The mother was exposed to radium nineteen months before the conception of this mongol boy. A small tumor, of doubtful malignancy, had been excised from the vulva, and the site was radiated, without shields. A severe burn followed. She was three months pregnant at the time, but the child, her first, was born at term, and is probably normal. Naturally I have wondered whether the radium could have affected the undeveloped ova to produce the mongol, and particularly whether at the present time (40 months since the radiation) such an effect on future pregnancies could still be expected. Recalling the known effects of radium and X-rays in producing mutations in seeds, etc., and the fact that in mongolism a defective ovum is known to be involved, makes one suspicious, at least. The bizarre aggregate of defects which characterize the mongol almost tempt one to regard mongols as mutations of the human species."

In like manner, there is little doubt that the "Catlin Mark" may and actually

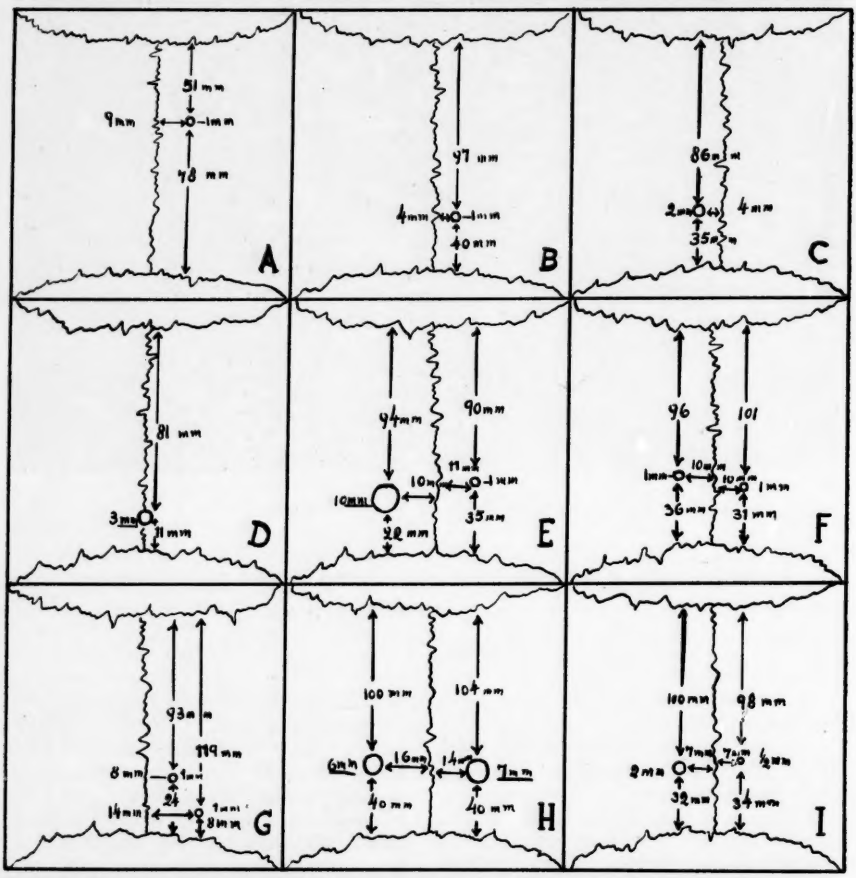


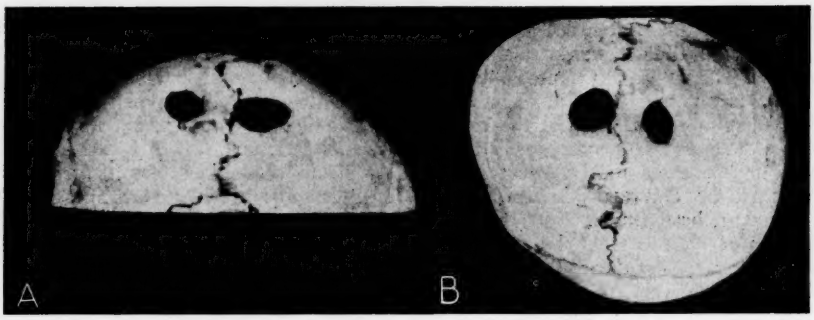
CHART SHOWING EXTREME VARIATION IN THE PARIETAL FORAMINA

Figure 11

Drawings show variation in location of foramina with distance to neighboring sutures. In some skulls both foramina are absent.

does arise as a mutation or as a sport* as is the case of numerous other hereditary characteristics. Assuming this conclusion to be correct, one would expect to find the defect hereditary in some families and in isolated individuals in other families, — as in the Indiana case. Since it seems highly probable that in common with many other mutant types, the "Catlin Mark" is continually appear-

*For convenience, the term "sport" is used to indicate a character which appears in a family line for the first time and then never appears again. Contrast with "mutation" which is transmitted as a mendelian character to later generations. In practice it is often difficult in human pedigrees to make such distinction clearly, because human progenies are so small that a true mutation might be classified as a "sport" according to the above definition, when a larger number of children and of grandchildren from appropriate matings would prove it to be inherited.



A CALVARIUM SHOWING A TYPICAL "CATLIN MARK"

Figure 12

The condition could well be mistaken for trephining, if it were found in a burial mound of Central America instead of in the dissecting room of Tulane Medical School. *A*—Direct posterior view. *B*—Viewed vertically to the plane of the fenestrae. (From Tulane University School of Medicine.)

ing in the human species both as a sport and as new mutations, as well as being transmitted in old established lines of inheritance, there is no reason to doubt but that this condition has existed for centuries, leaving thousands of human skulls "marked" with these strange head-top "eyes." Some races like the aborigines of the United States and Canada might have possessed less tendency to produce this striking variation. Such an assumption would account for the absence or scarcity of such "marked" skulls in certain regions.

Is the "Catlin Mark" a Mere Enlargement of Parietal Foramina?

The writer and most other investigators who have had an opportunity to make a study of prepared human skulls as well as of those of living cases having large paired openings in the parietal bones have concluded that such openings are merely "enlarged persistent parietal foramina", — through which pass the blood vessels which supply the scalp as will be shown presently. These parietal openings vary in size and position even from early embryonic stages. In some extreme cases of the "Catlin Mark" the openings were united in late fetal and early childhood stages so that the emissarium certainly passes through the abnormal opening. Whether or not ossification continues in such a manner as to form the characteristic "Catlin Mark" and also provide separate openings for emissary veins is problematical and certainly varies with individual cases. To find prepared skulls with a typical "Catlin Mark" and without the normal paired foramen near by, as is usually the case, would not be convincing evidence that the two large holes represent the paired parietal foramen, since any large collection of skulls will show cases with no signs of a foramen.

Pepper and Pendergrass[18,19] seem to take it for granted that parietal fenestrae are merely enlarged parietal foramina. They say, "It seems desirable to draw attention to the following facts: that the parietal foramina may be markedly enlarged; that such enlargement is a congenital anomaly with hereditary transmission; and that such large foramina can, during life, be discovered by palpation." These facts were observed and recorded by the author in 1922, and these openings were then considered merely as enlarged parietal foramina through which the small emissary veins passed.

However, observations of certain other workers indicate that the normal (but highly variable) paired parietal foramina and the anomaly known as the "Catlin Mark" (also variable) are not

one and the same thing, but have different origins, though anatomically they ofttimes (if not usually) fuse, or rather are superimposed. Dr. M. C. Sosman (Boston) in a discussion at the conclusion of Pepper and Pendergrass's paper, reports two cases in the Warren Museum at the Harvard Medical School. In one of these skulls, "just medial to the defect on the right *can be seen a normal parietal foramen*" through which the emissary vein passes. The other skull described by Sosman "also shows normal parietal foramina quite close to the midline." The condition shown in these skulls would tend to discredit the theory held by Pepper[10] and others that the large bilateral parietal foramina, known as the "Catlin Mark," are "enlarged parietal foramina." If these two unusual openings are merely enlarged foramina, the presence of both of the two normal foramina and also the two large holes called the "Catlin Mark" would mean a tendency toward bivalency of the foramina. If such a tendency existed, symmetrically bivalent, otherwise normal, foramina should not be uncommon. So far as the writer can ascertain, such a condition is rare.

Figure 10*A* shows a skull with what might be considered normal parietal foramina as illustrated in anatomy textbooks. However, almost any collection of a mere half-dozen skulls will show a decided variation, — all the way from complete absence of parietal foramina to large openings. The position of the foramina also varies. The skull pictured in Figure 10*B* shows only one foramen in an abnormal central position, in line with the sagittal suture.

The accompanying pen sketches (Figure 11) show the typical variations which might be expected in a collection of skulls. A survey of the skulls in a half-dozen collections showed wide variations in the position and size of the parietal foramina. Dr. H. S. French, Dean of the University of North Dakota School of Medicine, reports a typical collection. In 54 skulls of one group, 23 had no parietal foramina. Of the other 31 having foramina, only 14 showed the paired condition (Figure 11*A*, and Figure 11*F*) which is ofttimes spoken of as normal. Seventeen of the 54 skulls showed each a single foramen (Figure 10*B*, and Figure 11*A, B, C,* and *D*) with a great variation in size and position. Dr. Cummins of Tulane Medical College kindly submits additional data from Cave,[2] who cites the statistics shown below on variations of the "normal" foramina.*

In view of this tremendous variation and the large size of openings in certain skulls (Figure 11 *E* and *H*) which are unquestionably parietal foramina, one could easily conclude that the anomaly under consideration is in reality "enlarged parietal foramina." However, since certain skulls show contradictory evidence, we should refrain from designating this characteristic human anomaly as "enlarged parietal foramina" since this terminology presumes a definite understanding of the origin of the anomaly. With our present state of knowledge, such popular term as "Catlin Mark" of the present writer, or the more technical designation, "Fenestrae Parietales Symmetricae" of Halbertsma would seem more appropriate. It is probable that the "Catlin Mark" and the paired parietal foramina are conditioned by separate and highly variable genetical factors which determine their location. Since their positions are so nearly the same and both are variable, they may or may not both be present in the same skull, or in some individuals the two factors might be superimposed. Naturally, if a small hole (normal parietal foramina) and a large hole (the "Catlin Mark") should

	*Number of skulls	Both foramina present	Both foramina absent	1 foramen only	1 sagittal only
Greig	670	45%	30%	24%	0 3%
Cave	100 (Ancient Egyptians)	30%	29%	26%	11 %
Cave	150 (Modern)	32%	30%	22%	7 %

be superimposed, the boundary of the smaller would be lost in space. The extremely variable condition of these two factors, normally in about the same position in the parietal bones, would tend to substantiate this view.

Summary

1. The literature on the "Catlin Mark" is reviewed. This human anomaly is variously termed "Large bilateral parietal foramina," "Foramina parietalia permagna," or "Fenestrae parietales symmetricae."

2. Evidence indicates that the unusual bilateral fenestrae, anomaly of the human skull, are not merely enlarged parietal foramina.

3. This human anomaly has appeared for thousands of years, as shown by its presence in very ancient skulls.

4. When this skull anomaly (bilateral fenestrae in the parietal bones) is found in ancient skulls, it is erroneously classed as trephining.

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A Note on Terminology Regarding Intersexes

BEGINNING students of biology, and particularly those studying genetics, are often confused by the lack of uniformity in terms used to indicate various types of intersexes and related phenomena. For example, the word hermaphroditism is often used to indicate intersexuality in humans. It is also used for the common condition of sporophytes of plants in which both megaspores and microspores are borne on the same plants. Neither of these uses of the term is correct. Other much misused terms are "monoecious" and "dioecious". Even biological texts seem to assume that all species of flowering plants are either monoecious or dioecious when, as a matter of fact, most of them are neither. It is hoped, therefore, that the following outline may prove to be of some use in this connection.

(A) With regard to sexual organisms which show characteristics of both sexes in the same individual the following terminology would seem to be useful when correctly applied:

(1) A *hermaphrodite* is an organism (either animal or gametophyte plant) in which both ova and sperms are normally produced by one individual. Familiar examples of hermaphroditism are found in the common earthworm, many flatworms and the gametophyte generations of many mosses and ferns. The chief point to be stressed is that hermaphroditism

(*Continued on page* 314)

HEREDITARY MICROPHTHALMIA IN THE DOMESTIC FOWL*

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DURING the spring of 1940 a substantial number of chicks with bilateral microphthalmia were observed among those hatched from a pedigree pen of Barred Plymouth Rocks. It is not rare for poultrymen or research workers to discover an occasional chick with microphthalmia, especially of the unilateral type. For example, at this station unilateral microphthalmia has been observed in White Leghorns, Rhode Island Reds, and Barred Plymouth Rocks. On occasion these defectives have been reared to maturity and used as breeders, but the defect was not reproduced in later generations and it was assumed that these cases of unilateral microphthalmia were of non-genetic origin.

The hereditary bilateral microphthalmia reported in this paper (see Figure 13) is characterized by an eyeball with a diameter approximately one-half that of the normal. At hatching time the head presents a marked depression on each side in the region of the eyeballs. The eyeball is not visible on the exterior and the chicks are entirely helpless; at hatching they are usually found with their heads stuck out of the top of the pedigree basket like lighthouses. The microphthalmic gene also affects the structure of the comb, especially in the posterior region where a thickening is apparent. In many instances the comb was not only thickened but also doubled in the posterior region, and sometimes this doubling occurred along the entire length of the comb.

Hereditary microphthalmia has been observed in different mammals. King[1] found microphthalmia in rats to be inherited as a recessive with the interaction of several genetic factors. Hain[2] observed that microphthalmia in one line of albino rats was inherited, probably as a recessive. Eaton[3] observed a hereditary eye defect, including microphthalmia, in the guinea pig but was unable to establish a definite mode of inheritance. Bagg and Little[4] observed an eye abnormality, including the condition of greatly reduced eyeball, in mice, which was inherited as a Mendelian recessive. Verschuer[5] cites Waardenburg for the authority that microphthalmia in man is inherited as a recessive character. Several other workers report microphthalmia in man to be recessive, although it has also been reported as dominant, and still again, as sex-linked.

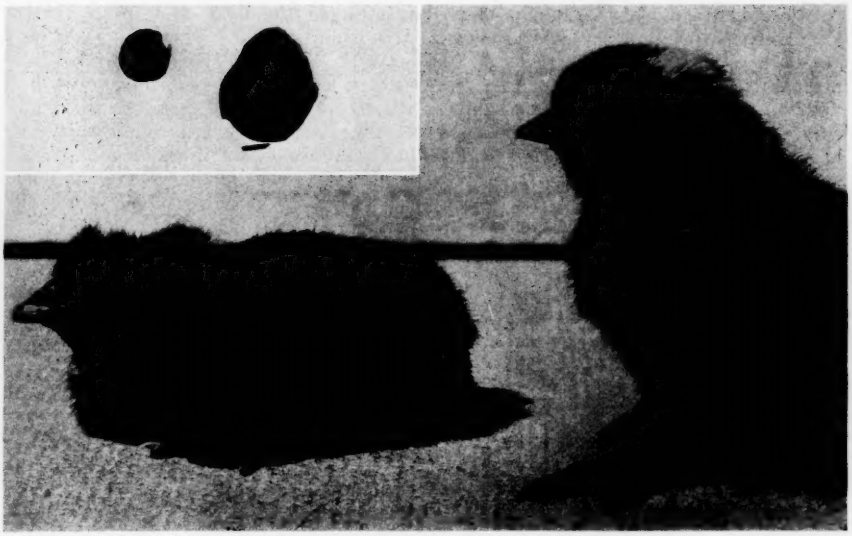
Landauer[6] reports that in the domestic fowl one of the manifestations of the creeper gene in the homozygous state is a microphthalmia.

Mode of Inheritance

The original mating from which microphthalmic chicks were obtained consisted of eight females mated to one male (H 1030). All the females were either full- or half-sisters to the male. Of the eight female breeders, four produced none but normal chicks, and four proved to be carriers of the microphthalmic gene. During the hatching season extending from April 4 to October 17 the ratio of normal to microphthalmic individuals produced by these carriers was 196:60. Assuming that microphthalmia results when a recessive gene occurs in homozygous form, and that heterozygous individuals present a normal appearance, it would be expected that the ratio would be 192 normal to 64 microphthalmic. Thus the calculated ratio agrees very well with the observed, which indicates that a single Mendelian recessive gene is involved. Detailed data are presented in Table I.

Since the defective chicks were unable to see to feed or drink it was not possible to rear any of them to maturity. Sev-

*Journal series paper of the New Jersey Agricultural Experiment Station, Rutgers University, Department of Poultry Husbandry.



"SMALL EYE" AND NORMAL CHICKS

Figure 13

Chick on left with microphthalmia; one on right is normal. In the insert at left are shown eyeballs, somewhat enlarged, from microphthalmic and normal chicks, respectively, to show relative size differences.

eral attempts were made at hand feeding, but none was successful. During the spring of 1941, however, several additional test matings were set up. The original male (H 1030), which was known to be a carrier, was mated with all the daughters of H 1700. Since H 1700 was known to be a carrier, it would be expected that two-thirds of her daughters would be carriers. Though the results are not so complete as they should be, nevertheless, the theory is not contradicted by the facts. Actually, eight daughters of H 1700 were reared to maturity, and of these three failed to produce any fertile eggs. Of the remaining five, three were carriers, and the other two produced none but normal chicks. From the three carriers (see Table II for details) were obtained 78 normal to 20 microphthalmic, whereas the calculated ratio would be 73.5:24.5, respectively. Again this closely approximates a 3 to 1 ratio, which further substantiates the theory that but one recessive gene is involved in the expression of this defect.

A second mating made in 1941 involved the crossing of the original female breeders (H 1603, H 1657, and H 1689) with a son of H 1657. The chances were two to one that this son would be a carrier, but he proved to be normal, because of the 157 embryos examined from this mating all were normal. Female H 1700 could not be used as a breeder in 1941 because of death early in that year.

The sex ratio of the microphthalmic chicks approached equality during both the 1940 and 1941 seasons and it is therefore concluded that no sex linkage is involved.

It should be added that in no instance was there any question as to classification. Embryos which died early could not be classified with certainty by macroscopic examination and therefore were not considered in the reported ratios. All others, however, could be classified with ease because of the absence of overlapping phenotypes.

Microphthalmic chicks did not hatch as readily as their normal sibs. For example, in 1940 the percentage hatch of normal embryos was 81 as compared with 43 per cent of the microphthalmic embryos. Again, in 1941 the percentage hatch of normal embryos was 68 as compared with 25 per cent of the microphthalmic embryos. Thus in respect to the percentage hatch, microphthalmia might be considered as a semi-lethal, and in respect to the early life of the chick, a true lethal gene.

The original pen of breeders possessed excellent egg production qualities. Eight full sisters of the male (H 1030) averaged 261 eggs per bird during their first laying year, ranging from 231 to 289 eggs per bird. The four female breeders that were carriers averaged 228 eggs per bird. This strain of Barred Plymouth Rocks was started in 1936 when day-old chicks were purchased from a commercial breeder. Inbreeding was not extreme until the 1940 breeding season.

Summary

Hereditary bilateral microphthalmia resulting from the action of a single Mendelian recessive gene has been observed in an inbred strain of Barred Plymouth Rocks. No sex linkage is involved. The gene reduces the diameter of the eyeballs by approximately one-half, and also has a thickening or doubling effect on the comb. This gene may be considered as a true lethal since all of the affected chicks died.

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TABLE I. Incidence of Microphthalmia in Four Families Sired by Male H 1030.

Dam No.	No. eggs		No. embryos dead early	No. embryos dead late		No. chicks	
	Set	Infertile		Normal	Microphthalmic	Normal	Microphthal.
H 1603	94	22	8	9	6	37	12
H 1657	125	18	15	8	15	66	3
H 1689	76	5	15	8	6	32	10
H 1700	90	28	18	12	7	24	1
Total	385	73	56	37	34	159	26

TABLE II. Incidence of Microphthalmia in Three Families Sired by Male H 1030. (1941)

Dam No.	No. eggs		No. embryos dead early	No. embryos dead late		No. chicks	
	Set	Infertile		Normal	Microphthalmic	Normal	Microphthal.
I 1219	61	41	0	2	3	15	0
I 1230	79	10	2	16	12	36	3
I 1234	20	5	4	7	0	2	2
Total	160	56	6	25	15	53	5

AN INHERITED DENTAL ANOMALY IN A JAPANESE FAMILY

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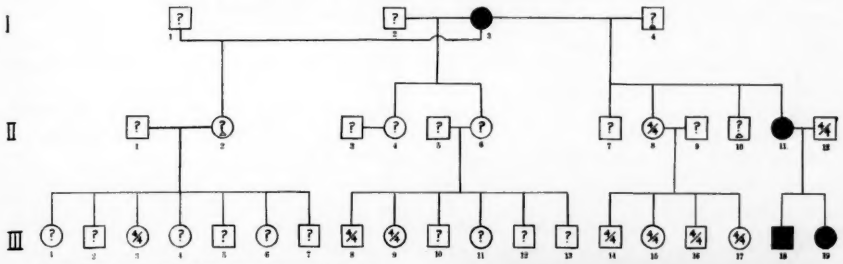

THREE GENERATION PEDIGREE

Figure 14

Pedigree chart showing inheritance of incisor deficiency (lack of one lower incisor) through three generations in a Japanese family. Solid squares and circles are males and females, respectively, exhibiting the incisor 4/3 trait. 4/4 denotes normal incisors. ? indicates that no information is available on dentition. Delta means deceased.

THE inherited dental anomaly here reported is the complete absence of one incisor in the lower jaw. It was discovered in a Japanese family in Honolulu, T. H. This trait may be expressed by the incisor formula 4/3 as contrasted with the normal 4/4. The X-ray photograph (Figure 15) reveals no evidence of the missing tooth. That the missing tooth is the outer left incisor is indicated by the fact that the outer right incisor has no antimere. The single tooth to the left of the mandibular symphysis is paired with the inner right incisor. So nearly centered is the visible portion of the middle incisor that it is impossible to determine, without the X-ray, whether this tooth takes root to the right or left of the symphysis. The rest of the dentition is apparently normal.

MISSING INCISOR

Figure 15

X-ray photograph of front teeth of lower jaw of III-19 (Figure 14) showing three incisors flanked by the canine teeth. The outer left incisor is absent.

The pedigree chart (Figure 14) indicates that the trait is transmitted as a dominant, most probably an autosomal dominant. Komai* published a large collection of pedigrees of various inherited abnormalities in the Japanese race including several involving the dentition and dental arches. Among these are cases of congenital absence of upper outer incisors and of premolars and cases of strikingly defective dentition in which as many as fifteen or nineteen permanent teeth are missing. He also reports cases

*KOMAI, TAKU. Pedigrees of hereditary diseases and abnormalities found in the Japanese race. 1934.

of abnormally large incisors of the upper jew and of conical incisors. In two pedigrees, the same gene has apparently determined the conical shape of the incisors in some cases and the complete absence of the same in others. Komai reports no cases such as ours of missing lower incisors. All the dental abnormalities reported by him behave as dominant characters in inheritance.

I am indebted to Mr. Hiroshi Daifuku (III-18), a student in the University of Hawaii, for collecting his family pedigree for me.

Summary

Here reported is a case of the inheritance of the absence of a lower incisor tooth through three generations in a Japanese family in Hawaii. The trait behaves as a dominant character in inheritance.

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Note Regarding Intersexes

(*Continued from page* 309)

is the normal condition in the species in which it occurs.

(2) A *gynandromorph,* on the other hand, is a genetic abnormality in which the individual, through chromosome loss or some other cause, abnormally develops certain male characteristics combined with female characteristics. In a sense, therefore, the organism is part male and part female. Gynandromorphism is found in humans, as is well known, and has been described in *Drosophila melanogaster* and in other species. It would seem impossible to determine in many cases whether the abnormality is due to genetic (chromosome) aberration but this would seem to be the most generally suspected cause. In certain cases, as where a sex-linked characteristic in one half the animal is absent in the other half, the evidence for chromosomal abnormality is very strong.

(3) Other abnormalities due to environmental conditions (either post-natal or prenatal) may have specialized names for the particular condition. Best known of these is, perhaps, the *free-martin* originally described by Dr. F. R. Lillie, in cattle, in which, due to prenatal anastomoses of the embryonic blood vessels of twins of opposite sexes, the male hormones have an effect on the development of the female. As described by Dr. Lillie, this effect produces abnormality of secondary sexual characteristics and sterility in the female animal which is now called a "free-martin." Comparable conditions may be found when gonads are diseased or atropied in various species such as the now famous cases of "sex-reversal" in fowls.

(B) In the spore bearing generation of higher plants (those showing heterospory) the following terminology would, if more strictly applied, make the problem much less complex to the student.

(1) *Monoclinous* plants are those in which both kinds of spores are borne on the same organ, that is, the same flower. Examples of this condition are found among our commonest plants, such as the members of the rose, the pea and the spurge families.

(2) *Diclinous* plants are those which bear the two types of spores in different flowers. In other words, they bear staminate and pistillate flowers separately. Diclinous plants are of two types, viz: (a) *Monoecious* plants are those in which the two types of flowers are borne on the same individual. A familiar example of this is seen in the common Indian corn (*Zea mays*) in which the tassel at the end of the stalk is an inflorescence of staminate flowers, while the familiar "silk" is the visible portion of the pistillate inflorescence. Another well known example of the monoecious condition is seen in the *Cucurbita,* such as the melon and the pumpkin.

(b) *Dioecious* plants are those in which the two types of flowers are borne on different organisms or plants. Familiar examples are the willows, the hollys and other common trees as well as many tropical plants such as the date palm.

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SIZE AND SHAPE OF HEAD HAIR FROM SIX RACIAL GROUPS

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IT has long been recognized that there exist racial differences in hair form, but it is only recently that the great range in variation that exists within a single race has been demonstrated. Danforth[1] remarked that hairs growing side by side may vary in their index by 30 or more points and that the index of hairs from 25 white persons ranged from 50 to 89. Trotter[5] states that the greatest range in indices probably is present in the white race, ranging from 47 to 92. Woodbury and Woodbury[7] showed much overlapping in hair form and texture between several Indian tribes, although in many cases real tribal differences existed. On the other hand, Kneberg[2] has attacked the hair index as a racial criterion because of its great variability and lack of conformation with hair form. The main objection to be made to some of these and many of the earlier works has been the inadequacy of the samples. Now that an improved technique has been devised, statistical analyses are available for hair measurements, and variations found between races and between individuals of any one race can now be adequately coped with.

This paper is the result of the application of a new technique to human hair sectioning described recently by Steggerda[3], and a continuation of the study outlined by Steggerda and Eckardt.[4] The method is described fully in the former paper and it need only be said here that it enables the sectioning of at least 100 hairs at one time.

Materials and Methods

The hair of the six racial groups herein considered was collected by the senior author on his annual field trips in the United States and Mexico. The Dutch whites are children residing in or around Holland, Michigan; the Hopi, Navajo, and Zuni samples were collected in Arizona and New Mexico at various government schools; the Negroes are from the area of Tuskegee Institute, Alabama; and the Maya, from Piste, Yucatan. The individuals range in age from 9 to 19 and average around 14 or 15 for all races. The hair was cut close to the scalp in the occipital region of the head in all cases, since hair is said to vary in size according to the region of the scalp.

When brought to the laboratory the samples were washed and sectioned and mounted on slides. The cut was made 20 mm. from the base. Camera lucida drawings of the sections were then traced on thin paper. The scale of an ocular micrometer was likewise traced and the readings etched permanently on a piece of celluloid; the hair outlines could then be measured directly by this transparent scale. From 70 to 100 hairs from ten individuals, consisting of five males and five females, of each race were measured.

The area of the hair section was at first measured with a planimeter and the units then converted to square millimeters. However, it was found that for hairs whose outlines were not too irregular, a method of using the area of a circle was accurate enough to justify its use, especially since for small areas the planimeter readings were rather inaccurate. The formula so used was $A = \pi \left(\frac{D_1 + D_2}{4}\right)^2$ where D_1 is the maximum diameter and D_2 is the minimum. The agreement between this formula and the converted planimeter readings are as follows:

planimeter --	.004938	.003918	.005079	mm.^{2}
formula —	.004899	.003895	.005109	mm.^{2}

A scale was then made whereby from the sum of the two diameters, the area could be read off directly.

Interracial Variation

Table I gives an indication of the range of values that may occur in one racial stock. While the other races con-

sidered in this paper are not quite so variable, the Negroes have been selected to emphasize this important point. Area ranges from 28.4 (square units)* to 50.9, a difference of 22.5; the range in maximum diameter amounts to 31 micra, and the greatest difference in index amounts to 17 points. Intraracial variation in dimensions can easily exceed interracial differences. It is therefore apparent that many hairs from a good sample of individuals are necessary to arrive at a proper mean value for any one race, and since the hair from one individual has a much greater range in size and shape, the same consideration must be given to samples from one individual head.

Sex Differences

The data in the literature on sex differences in hair size and shape are not very conclusive, very likely a result of insufficient material and great variability of individual hairs. Trotter[5] found that males had a larger index than the females for the age group 10-19, although her differences were not great enough to be significant. In the French Canadian series Trotter and Dawson[6] found that the opposite was true for the 10-14 age group, while in the 15-19 age group the index was again greater in the males. These results were not significant and are accounted for by Dr. Trotter as chance variations due to small numbers. Woodbury and Woodbury[7] claim to have found no sex differences, and since their data are lumped, no comparison is possible.

Our data, however, show very definitely that the males have rounder hair than the females for the age group considered. This is true for all the races and the differences are statistically significant except in the case of the Dutch.

In four races the males have a larger area than the females and in three of these the differences are significant; on the other hand, in only one of the races whose hair is larger in the female is the difference significant. Trotter[5] found no sex differences in hair area; Trotter and Dawson[6] found that the females had a larger area in the 10-14 age group, but reversed in the 15-19 group. None of these differences, however, were significant.

There are no consistent sex differences with regard to maximum diameter; the males have the largest diameter in two-thirds of the races, the same ones wherein the males had the greater area. In both cases, the Hopi males exceed by only a slight margin. Except for the Dutch, the males exceed in minimum diameter and in all races but the Zuni, the differences are significant.

As determined by the coefficient of variation, the females show a greater variability in index than the males except in the case of the Dutch. Trotter, and also Trotter and Dawson's figures show a similar greater variability for females in the ages 10-19. However, none of the other three measurements reveal any consistent relation to sex.

Racial Differences

From the means for the index in Table I, it is apparent that the Maya have the most nearly circular hair, and the Negroes the most elliptical. Although the indices drop rather gradually in the first five races, the differences between any two consecutive races is more than three times the probable error of the difference (except between Hopi and Navajo) and is therefore probably significant. The great drop in index between the Dutch and Negro is of the magnitude of 46 times its probable error of the difference.

TABLE I. Means of Cross Section Measurements of Negro Hair from Ten Individuals.

Index	Maximum diameter	Area
♂ 58.25 ± .88	90.04 ± 1.15	43.59 ± 1.05
♂ 66.00 ± .69	94.17 ± 1.40	47.92 ± 1.20
♂ 56.97 ± .64	102.89 ± 1.49	50.96 ± 1.27
♂ 59.21 ± .53	95.59 ± 1.05	45.27 ± .84
♂ 66.34 ± .49	74.91 ± 1.24	30.86 ± .86
♀ 52.32 ± .62	80.61 ± 1.27	28.45 ± .73
♀ 56.71 ± .76	82.01 ± 1.41	31.46 ± .88
♀ 54.92 ± .68	88.72 ± .98	46.55 ± 1.13
♀ 49.35 ± .85	105.96 ± 1.34	35.41 ± .66
♀ 59.55 ± .72	96.38 ± 1.52	48.08 ± .94

*Square units equal square micra divided by 100. This value was chosen to obviate cumbersome figures.

TABLE II. Means, Standard Deviations, and Coefficients of Variation, with Probable Errors, for Head Hairs of Six Racial Groups.

Race	Males N	Mean	S.D.	C.V.	INDEX Females N	Mean	S.D.	C.V.	Total N	Mean	S.D.	C.V.
Maya	482	85.04 ± .33	10.6 ± .23	12.4 ± .27	504	80.93 ± .35	11.6 ± .25	14.3 ± .31	986	82.94 ± .24	11.3 ± .17	13.7 ± .21
Hopi	283	82.98 ± .43	10.8 ± .30	13.0 ± .37	334	78.09 ± .51	13.9 ± .36	17.8 ± .48	617	80.89 ± .36	13.2 ± .25	16.3 ± .32
Navajo	500	82.53 ± .34	11.2 ± .24	13.5 ± .29	502	76.94 ± .41	13.6 ± .29	17.6 ± .39	1002	79.73 ± .27	12.7 ± .19	16.0 ± .25
Zuni	321	80.46 ± .45	12.0 ± .32	14.9 ± .40	322	73.95 ± .53	14.0 ± .37	18.9 ± .52	643	77.19 ± .36	13.5 ± .25	17.4 ± .34
Dutch	360	76.12 ± .52	14.5 ± .37	19.1 ± .50	498	75.90 ± .38	12.4 ± .27	16.3 ± .36	858	75.99 ± .31	13.4 ± .22	17.6 ± .29
Negro	361	61.54 ± .32	9.1 ± .23	14.8 ± .38	512	54.48 ± .36	12.1 ± .25	22.2 ± .49	873	57.40 ± .26	11.5 ± .18	20.0 ± .34
AREA (Square Units[1])												
Hopi	283	45.74 ± .61	15.3 ± .43	33.5 ±1.05	334	44.52 ± .76	20.6 ± .54	46.2 ±1.44	617	45.08 ± .50	18.4 ± .35	40.8 ± .90
Zuni	321	42.24 ± .76	20.1 ± .54	47.5 ±1.52	322	44.51 ± .61	16.2 ± .43	36.4 ±1.09	643	43.37 ± .49	18.3 ± .35	42.2 ± .92
Maya	482	43.17 ± .43	14.0 ± .30	32.4 ± .77	504	39.02 ± .39	12.9 ± .28	33.1 ± .78	986	41.05 ± .29	13.6 ± .21	32.2 ± .54
Navajo	503	43.63 ± .56	18.7 ± .40	42.6 ±1.06	492	37.11 ± .48	15.8 ± .34	42.6 ±1.07	995	40.65 ± .38	17.6 ± .27	43.2 ± .77
Negro	361	43.35 ± .53	14.9 ± .37	34.4 ± .96	512	37.74 ± .46	15.4 ± .33	40.8 ± .99	873	40.06 ± .35	15.0 ± .25	38.7 ± .71
Dutch	360	22.50 ± .35	9.7 ± .24	43.3 ±1.28	498	25.29 ± .32	10.5 ± .22	41.5 ±1.13	858	24.11 ± .24	10.3 ± .17	42.7 ± .81
MAXIMUM DIAMETER (Micra)												
Negro	361	91.03 ± .66	18.5 .47	20.4 ± .53	512	90.33 ± .65	21.9 ± .46	24.31 ± .54	873	90.62 ± .47	20.6 ± .33	22.7 ± .39
Zuni	321	81.18 ± .82	21.8 ± .58	26.8 ± .76	322	87.47 ± .80	21.3 ± .57	24.3 ± .68	643	84.33 ± .58	21.7 ± .41	25.8 ± .51
Hopi	283	83.86 ± .71	17.7 ± .50	21.0 ± .62	334	83.57 ± .85	23.0 ± .60	27.5 ± .77	617	83.70 ± .56	20.7 ± .40	24.8 ± .50
Maya	482	80.55 ± .48	15.7 ± .34	19.5 ± .44	504	78.54 ± .48	16.0 ± .34	20.4 ± .45	986	79.53 ± .34	15.9 ± .24	20.0 ± .31
Navajo	500	80.95 ± .62	20.5 ± .44	25.3 ± .57	502	76.57 ± .54	17.9 ± .38	23.4 ± .52	1002	78.76 ± .41	19.4 ± .29	24.6 ± .49
Dutch	360	61.53 ± .60	16.8 ± .42	27.2 ± .73	498	65.67 ± .47	15.4 ± .33	23.4 ± .53	858	63.93 ± .37	16.1 ± .26	25.2 ± .43
MINIMUM DIAMETER (Micra)												
Hopi	283	68.32 ± .29	7.3 ± .21	10.6 ± .30	334	62.77 ± .51	13.8 ± .36	22.0 ± .60	617	65.31 ± .35	13.0 ± .25	19.9 ± .40
Maya	482	67.46 ± .36	11.6 ± .25	17.1 ± .38	504	62.45 ± .29	9.8 ± .21	15.7 ± .35	986	64.90 ± .24	11.0 ± .17	16.9 ± .26
Zuni	321	63.41 ± .55	14.5 ± .39	22.8 ± .64	322	62.22 ± .39	10.3 ± .28	16.5 ± .45	643	62.81 ± .33	12.6 ± .23	20.0 ± .39
Navajo	500	65.73 ± .43	14.3 ± .30	21.7 ± .48	502	54.47 ± .33	11.1 ± .24	20.4 ± .45	1002	61.96 ± .28	13.4 ± .20	21.6 ± .34
Negro	361	56.66 ± .37	10.4 ± .26	18.3 ± .47	512	48.21 ± .33	11.2 ± .23	23.3 ± .52	873	51.70 ± .27	11.7 ± .19	22.6 ± .38
Dutch	360	45.39 ± .33	9.3 ± .23	20.5 ± .54	498	48.65 ± .29	9.5 ± .21	19.6 ± .43	858	47.28 ± .22	9.6 ± .16	20.3 ± .34

In comparison, Woodbury and Woodbury found slightly lower values for the hair index of Navajo and Hopi, being 77.25 ± .87 and 78.81 ± .94 respectively. The discrepancy between these values and ours is not sufficiently great to be outside the realm of chance variation. Our Dutch white series compared with Trotter's[5] American whites of the same age group (10-19) show a difference of about 2.3%. Also our Dutch have a slightly greater index than Trotter and Dawson's French Canadians of the same ages: 75.9% compared to 73.1%. Again these data on whites compare rather favorably with ours.

In general it appears that variability decreases as the mean index increases and the Negroes with the lowest index, have the highest coefficient of variation. The reverse is true for the Maya. If this relationship is a good one, it would be expected that the females would have greater variability than the males; this has already been shown to be the case.

The Hopi have the largest hair area of all the races, almost twice that of the Dutch who have the smallest. Woodbury and Woodbury considered hair area by averaging the maximum and minimum diameters; this they called the hair's "texture." Among the Indian tribes that they studied, it was found that the Hopi had the coarsest hair. Among our Indians and the Negroes, the differences in hair area are gradual, not exceeding 2.3 square units, but there is a drop of 15.9 square units between the Negro and the Dutch.

The Negroes have the largest hair diameter of all but have a very small minimum diameter, a fact that accounts for their low index.

From these figures dealing only with cross section material it appears that the Indians resemble each other closely from the standpoint of index and area. The Negroes have an area that is equal to that of Indians but the hair differs strongly in shape, whereas in the whites the index almost approximates that of some Indians yet the size as indicated by area and diameter is much smaller.

Summary

Because of the great variation in size and shape of human head hair, it is necessary to have a large number of hairs from each of several individuals in order to arrive at a fair evaluation of hair dimensions. Intraracial variation can exceed interracial differences.

For the age group considered, the males have a higher index than the females for all six races, and this index is more variable in the females. No other sex differences consistent for all six races was revealed.

The Maya have the roundest hair, and the Negroes the most elliptical; the Hopi have the coarsest hair and the Dutch, the finest. The cross sectional hair dimensions indicate that the Indians, the Dutch, the Negroes fall into three separate categories, the former distinguished by large, round hairs, the second by small intermediate shaped hairs, and the latter by large hairs with elliptical cross sections.

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THE ROLE OF PSYCHOLOGY IN EUGENICS

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THE word "eugenics" has a dual meaning, depending on whether we consider the subject as science or practice. The original Galtonian definition is concerned with the study of all the agencies under social control which may improve or impair the inborn qualities of the future generations of man, either physically or mentally. Or, in other words, the study of factors in heredidy and environment which may affect human development, as well as those which may affect differential reproduction of varied genetic types. The laws or principles contributed by the social and biological sciences, selectively organized, are the basis of eugenic wisdom.

Eugenics, as an applied science, or movement, or program, is concerned with utilizing these laws and principles, directed toward the goal of the eugenics ideal, namely the improvement of human quality. In its applied aspects, eugenics can be compared with medicine. In both disciplines, students are trained in an organized circle of knowledge pertinent to a particular field of service. This special wisdom is then applied with what art, skill, and technical proficiency the practitioners can muster to produce desired results in the way of better health and development.

Eugenics, like medicine, stresses the preventive functions of its work, the positive even more than the negative steps to be taken to insure human well-being and health and soundness of mind and body, particularly for future generations. Needless to say, both disciplines draw largely from the same scientific areas for information and knowledge; and also for principles and practices in personal application and public education.

The sciences which contribute most extensively to the fund of eugenic wisdom are psychology, biology, sociology, anthropology, economics, and the new science of demography, — the study of population trends. From social demography, eugenics derives its understanding of the social consequences of changing trends in birth rates, death rates, and rates of natural increase within the country, including the differential fertility of groups, classes or sections within a national population. Eugenics crystalizes the findings of sociology and anthropology with respect to the influences of culture and custom on human behavior and personality and on human reproduction. From economics, eugenics learns of hazards and inducements to parenthood governed by maternal needs. From biology, it learns of the laws of inheritance and transmission of traits — particularly physical traits — and those factors concerned with maintaining bodily health and well-being which affect the quality of man. From psychology, eugenics derives insight and understanding of the qualitative aspects of population, especially those psychological characteristics of intelligence and personality which, far more than the physical, will determine the ultimate trend of the race. From psychology, also, eugenics has assembled and organized contributions to a better understanding of the hereditary-environmental influences governing the appearance and development of intelligence and personality. Particularly is eugenics concerned with individual differences in populations; also with group differences when their basic factors can be traced out by scientific study.

Two Kinds of Eugenics

Thus derived and thus oriented, applied eugenics can be considered to fall in two categories: clinical eugenics and social eugenics. The former would approach the eugenic problems of the individual person or couple or family line; the latter, the eugenic problems of a group or nation. In both individ-

ual or group application, the eugenic goal of human improvement will guide activity. In both areas, techniques for enlightening the client or the public will be drawn from acceptable principles and practices already found useful and sound in such other professional fields as: individual counseling in a medical or psychological clinic or private practice; in teaching, writing, social and community service, public health, education, and publicity in general.

To the psychologist who is familiar with the origin of psychological clinics for the adjustment of problem individuals, it is not surprising that the eugenics movement also began its work with an emphasis on negative aspects. The continued reappearance within family lines and society in general of inferior or inadequate individuals who were a problem to themselves, to their families and to the state, first turned public attention to alleviative measures.

It was within the powers of almost any observer to convince himself that certain physical defects, such as club foot, absence of fingers or limbs, or blindness, deafness and other abnormalities, were transmitted from one generation to another through the germ plasm. From this it was not a far jump to deduce that such physiological conditions as are to be found in disease or mal-functioning of organs could also be passed on genetically. Stomach ulcers, the tendency to have cancer or diabetes or tuberculosis, were also seen to follow family lines. Similarly it was noted that individuals of one family stock presented more than a random chance showing of such psychological defects as mental deficiency and mental disease. Elaborate genealogies were drawn up to present evidence of the transmission of these defects from ancestors to descendants, and the not unexpected conclusion was drawn that these conditions, too, were inherited.

The degree to which the manifestation of inherited tendencies might be dependent upon environmental factors was not at first recognized. With the coming of scientific research studies in this field — carried out largely by psychologists — the differential role of these two variables, heredity and environment, was better understood. The eugenics associations were among the first to utilize and to sponsor studies in a further clarification of this controversial question. Today, eugenics is actively engaged in promoting research in other areas of direct concern to, or even basic to, its field of activity, for only on scientific evidence will eugenics erect its own edifice and program.[2]

In the beginning, however, eugenics had to proceed on the material which was currently available. It was thought — and the eugenists themselves were among those promulgating the idea — that many forms of social inadequacy which reappeared through successive human generations were necessarily passed on as innate biological characters *via* the germ cells. Among the deficiencies and defects noted for their reappearance through successive human generations were criminality and delinquency, chronic pauperism and dependency, alcoholism, drug addiction, sex perversion and moral violations. At that time, eugenics programs centered negatively around the reproductive deletion from the body public of those manifesting such defective or low-grade potentials. Much was said about birth control and sterilization of the "unfit": so much indeed that the two terms "sterilization" and "eugenics" became in the public mind almost synonymous.

But eugenics is more than an argument for eradicating the unfit; it is also a force for promoting the greater reproductivity of good biological stocks. People who were in doubt as to which type they, individually, might represent (as could easily happen when both good and bad characters emerged within the one family line) felt a great need for enlightened counsel in the science of human genetics. This was the kind of service which originated and for many years was fostered at the Eugenics Record Office, a part of the Carnegie Institute of Washington, at

Cold Spring Harbor, N. Y. The work done there in assembling needed data from the clients who applied for guidance with respect to problems of their own marriage and reproductivity was an excellent beginning of what might later be called a eugenics clinic. That it was not so called may be due to the fact that it was directed by a biologist rather than a psychologist.

Clinical Eugenics

Clearly, the basis for a good working practice in clinical eugenics has already been set up. The motive for visiting such a clinic will generally be to ascertain probable consequences of a proposed marriage; or, in psychological terminology, to seek an adequate adjustment for an individual, or couple, or family, with respect to problems of reproductivity for the family lines concerned. Preferably, this eugenic guidance should be sought before unions are consummated; or, if a birth has already taken place, of a sort to suggest abnormality in a family line not previously suspected of genetic weakness, then eugenic guidance should be drawn upon before other conceptions occur. In this day of rather efficient contraception, unwanted births — at least to those parents who are of average or better intelligence — are no longer to be regarded as a visitation from providence, to be borne with resignation and grace. The problem today is rather one of deciding whether or not a contemplated pregnancy will carry with it a hazard or a promise of fulfillment in the birth of superior offspring.

The procedures or techniques of a eugenics clinic will include:

1. A genetic investigation of the clients and their families.
2. A medical examination of the clients, covering both partners to the proposed union.
3. A psychological examination of both.
4. Developmental records, case histories, and an analysis of the adjustment of the clients to life.
5. An analysis of the findings; presentation of the evidence to the clients, with discussion, evaluation, and the best possible prediction as to what can be expected in the progeny of the proposed union.
6. Record-keeping and research.

Since the propagation of a new life will normally be a function of two family lines, a eugenical analysis will be directed at its best to a genealogical study of the relatives of both partners to the impending union. The genetic investigation of the families will be conducted by specially trained eugenic social workers, whose business it will be to gather material on all the relatives, ascendants, descendants, if any, and collaterals for two or more generations preceding the propositus, with special study of his siblings and previous offspring, if any. It is best for couples to present themselves as a unit, but it may well happen that only one member of the proposed partnership is intellectually or emotionally concerned about his genetic potentials, and that the other does not appear for study or advice at the clinic. In all cases, the missing partner should be urged to come, not only to participate in the discussion of the data gathered but also to contribute information as to his own line. Genetic strength in one branch of the union may be enough to outweigh genetic weakness in the other, in which combination, the probable prediction for offspring would be different than if limited to one partner alone.

Data accruing from the medical and psychological examinations and the rather thorough case histories of both partners are a necessary adjunct to good clinical eugenics. The degree and nature of the material to be selected for discussion will depend in part on the mental capacity of the clients to grasp it and its implications for the progeny. The kind of counsel to be given clients will also be determined in large part by the many variables which enter into the analysis. Each couple or case unit will be highly individualized as to recommendations. For instance, in those cases which show no serious degree of maladjustment in their personal case histories, but who may show serious genetic weakness — perhaps on both sides — the recommendation may be marriage, but without offspring. Or to

partners, one or both of whom are suffering from some form of secondary mental defect, a similar recommendation may be made. In the first case, the proposal is offered to protect the offspring from hereditary defect or injury; in the second, to protect the offspring from the definite environmental injury which can come to a genetically normal child who is reared by feebleminded parents, too poorly equipped mentally to give him the kind of parental guidance every child ought to have. In both cases, marriage without offspring can bring satisfaction and happiness to the couple, without risking the welfare of children.

To take another example of the type of problem which may appear in a eugenics clinic, consider the case of partners whose genetic, developmental, medical and psychological examinations are excellent. To partners of this caliber, the eugenic counselor can well point out the desirability of reproducing a large family, at least six children to insure that the two excellent lines will be more than replaced in future.

With partners, for whom both the genetic and psychological evidence is poor or doubtful, marriage with contraceptive help, may be indicated. In cases of very poor genetic and psychological quality, marriage with sterilization may be suggested, provided a degree of marital adjustability can be assured for the sake of compatability. When developmental and psychological data are good, but the genetic prospects are doubtful or poor, then marriage, but with a limited number of children — for example, two — may be the suggestion, less than the replacement quota of about three per couple.

In addition to the reproductive problems for which the clients seek advice, these examples will strongly suggest the kind of human marital adjustment problems which appear in almost any adult psychological clinic. Clinical eugenics accepts gladly these contributions from clinical psychology.

In the eugenics clinic, the counselor will not seek to dominate the decision for the couple or the individual seeking help. He will present the evidence, discuss its significance, indicate the mathematical probabilities of deficiencies or defects in the offspring, point out a course, and leave the decision to the client and his family. Eugenic service of this sort is advisory, voluntary, and at no time is the advised course to be compulsory. Only this kind of eugenics can be tolerated in a democracy.

Throughout the eugenics movement in America, the emphasis is upon the rights of the forthcoming generation; the rights of children to opportunities for normal development everywhere; the right to be born to parents who will care for them properly and wisely; the right to be born free of hereditary defect or abnormality. Any psychologist will recognize in this set of principles a close kinship to the practices of a good psychological or educational clinic.

As in all good clinics, also, case records should be systematically compiled and filed. The forms should be framed with regard to research needs later, for in the eugenic clinical area, as in most others, much more reliable insight is required if service is to improve with the years. It devolves upon the eugenics workers to assemble materials, accruing from their contacts with clients, in such a way that these will aid in building up further understanding of the genetic factors underlying human psychological and physical traits. Preferably, also, a system of follow-up cases should be included, in reference to the subsequent adjustment of the clients within marriage, and particularly should details be gathered and filed about their offspring and in turn of the later adjustment of these children. Only by such complete returns can science advance in this so complex and highly important area of clinical service.

Social Eugenics

Whereas clinical eugenics centers chiefly around the reproductive adjustment of the individual family, social eugenics is concerned with awakening in the whole public a point of view and

in bringing enlightenment to a nation about the very problems which confront the workers in the clinic. A fundamental principle of social eugenics is that inadequate adults should realize the desirability of not having too many offspring; and that adequate adults should more whole-heartedly accept their responsibility and their privilege in contributing generously to a better population. A rational social eugenics aims at lowering the reproductivity of individuals who are below the average in important traits of health intelligence, and personal adaptability. Even more important, it seeks to maintain in those who are above average in these traits, a replacement rate sufficient to insure that their progeny will progressively increase with each subsequent generation. In so far as ignorance, poverty, and general inability to use contraceptives are responsible for the greater fertility of the sub-average couples, social eugenics is interested in a wider and more effective dissemination of family-limitation methods, and to this end, encourages the establishment and services of birth-control clinics throughout the country. These are needed in sparsely settled and culturally deprived rural areas as well as in submarginal city slums, where poverty *per se* has already led to their existence.

In so far as economic handicaps (arising out of the complexities and inequalities of income and expenditures) may prevent adequate and superior parents from having larger families than they are now having, social eugenics is promoting the idea that public compensations should be set up which would tend to offset these handicaps. In the last analysis, although it is the parents who actually bear the children, it is the public, the state, and the larger world which profit from their coming into life. The obligation of the community to share the expenses, sacrifices and inconveniences which attach to the bearing and rearing of children is a just one, which it can be taught to accept.

It is important for a nation to understand the trend of its population, both as to numbers and as to quality. Thus social eugenics must be concerned with disseminating information about the differential fecundity of the various sections of the population. The balance between birth rates and death rates in different groups within the whole, the factors that make for survival or decline in different regional, racial, and socio-economic groups, are basic in the growth or decline of nations. The vast importance of these factors must be understood by the people of a democracy.

As a part of this quantitative aspect of population study, social eugenics seeks to find out why different types of people regulate the size of their families in the way they do. It is especially concerned to analyze the objections of superior or above-average parents who too rigidly limit the number of their children. Social eugenics seeks to make large families a popular cultural concept among those who are above average in their capacity to respond to environmental opportunities, just as it has sought to make the concept of a smaller family acceptable to those whose responsiveness to environment is limited.

As a part of the qualitative study of population, social eugenics seeks to understand all the environmental factors, physical and social, which affect child development. To this end, the excellent studies in child psychology have been heavily drawn upon. Social eugenics will endorse or promote the work of agencies which teach mankind how to make a better adjustment to the world it is to live in. For example, social eugenics has allied itself with the work of the visiting nurse, the recreation associations, the day nurseries, social service and social welfare centers, the forces of education and the church.

Chiefly, however, social eugenics wishes to assemble and use the laws of inheritance of traits, particularly of mental inheritance, to bring about a better selection of family stocks, based on hereditary selection. Scientific knowledge of mental inheritance is largely the contribution of research psychology. Social eugenics seeks to apply the

known laws of human heredity by identifying carriers of proficiency as well as of deficiency. To that end, it recognizes fully the interlocking role of environment with heredity. Only by equating the environment can the individual differences (or group differences) due to heredity be made to stand out. When the inequalities of environment have been evened up to a greater extent than they are at present, it is recognized that certain families, whose development was previously thwarted, will have a better opportunity to express their innate capacities. In an equated and superior environment, those family lines whose retardation was once due to environmental restriction will respond to changed conditions and thereby be recognized as carriers of good genetic capacity.

But already, in spite of present environmental inequalities and handicaps, many individuals have demonstrated their capacity to adjust reasonably and well to life. Social eugenics takes the position that such persons can now be designated as having good genetic capacity on the principle that a trait needs but to manifest itself to prove that it exists. Such persons whose reactions are of positive social significance should be encouraged to have enough children more than to replace themselves. Families, on the other hand, who have not yet been able to rise above the average even when given assistance, should be able in a generation or two to demonstrate their better potentials where innate superiorities are actually latent, thus differentiating themselves from others which, even with the better environmental help, are still not able to rise even to average performance. Thus, in time, when environments are no longer so unjustifiedly diverse as at present, the still sub-average stocks will be recognized as carriers of generally inferior genetic capacity. It is to be hoped that they will more conscientiously refrain from replacing themselves in future generations—provided, of course, that they accept the eugenic philosophy promulgated today.

If the social philosophy which is taking shape in our society today becomes a working principle for bringing the more abundant life to all, such differences as will be found among a people will progressively cease to be dependent not upon unevenness in the environment but will increasingly depend upon differences in hereditary quality. Thus in the future, when the environment for all citizens has been lifted to a high level, the role of heredity will be more significant than the role of environment in making people different. Under such conditions heredity will play a larger part in explaining differences than at present, because today the origins of differences are still obscured by complexities of both environment and heredity. Thus the eugenist sees his active interest in unraveling the interlocking effects of these two variables enormously advanced by social measures to equalize the nurture factor.

The evening up of environmental inequalities has been advocated as an expression of social justice, but it is also an excellent research technique for isolating genetic variables. When good lines are identified, eugenics will always encourage the fullest measure of reproductivity for these lines. The eugenic movement will then be much better equipped than it has been in the past to supply materials and guidance for the work of the eugenics clinic. As that time approaches, we can expect a more active promotion and fostering of clinical eugenics. Today the stress is on public enlightenment and paving the way.

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EUGENICAL IMPLICATIONS OF RECENT STUDIES IN THE FIELD OF MENTAL DEFICIENCY

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IT is heartening to discern within the present-day darkness of social ignorance and failure, the dim but growing awareness that human subnormality is a major problem of social welfare. Despite this awareness, progress in the social control of human subnormality has been hindered by wide diversities in concept and practice, by lack of agreement as to the essential features of deficiency, and by an absence within this field of a satisfactory basis for coordinating the work of the medical and social sciences. However, there are scientific and social forces which tend to unify the work in the field of human subnormality. Especially promising is the interest of present-day genetics and eugenics — fields of research and practice, which, like the study of subnormality itself, have implications for the biological, medical and social sciences, implications which cannot be restricted to any one single discipline. A review of recent studies showing the contributions which already have been made to the problem of human subnormality may indicate ways in which genetics and eugenics can provide the basis for coordinating the medical and social approaches to this problem.

Studies in Clinical, Experimental and Social Genetics

Current studies of genetics and eugenics which are of special significance for the field of human deficiency may be roughly grouped under three headings— "clinical" genetics, "experimental" genetics, "social" genetics.

Clinical or medical genetics in relation to mental deficiency includes studies of *specific organic deficiencies,* of *familial deficiencies,* and of *genetic deficiencies in relation to mental illness.*

A. Clinical or Medical Genetics

1. *Specific organic deficiencies.* — These may be always or only occasionally associated with mental deficiency. Genetic deficiencies found occasionally associated with or existing independently of mental deficiency are, however, equally manifestations of a basic organic defect and might thus be also included in the general field of human deficiency. The most extensive review and study of inherited biochemical deficiencies found associated with mental deficiency has been made by Jervis[1] who lists among these deficiencies phenylpyruvic oligophrenia, which is an inborn failure of the body (determined by an autosomal recessive gene[3,4]) to oxidize phenylpyruvic acid[2]; amaurotic idiocy (Tay-Sach's Disease), which is caused by a genetically determined inability of the body to oxidize the lipoid sphyngomyelin; certain inherited degenerative basal ganglion lesions; albinism — which is biochemically related to phenylpyruvic oligophrenia; glycogenosis — a genetically determined inability properly to metabolize carbohydrates; and, tubero-sclerosis, in which a genetic factor is probably operative. Jervis has stated that "In the majority of cases, both mental defect and metabolic error are correlated expressions of the same abnormal process, the nature of which is a matter of future investigation."

Additional hereditary organic deficiencies associated with mental deficiency are represented by: hemiplegia, naevus and mental defect described by Brushfield and Wyatt[5]; bilateral anophthalmos and mental deficiency by Kallman, Barrera and Metzger[23]; microphthalmia and mental deficiency (Fraser-Roberts[12]). Most specific organic deficiencies with

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hereditary background have been described largely in the low and middle grade defective groups.

In investigating organic deficiencies with hereditary background, it is especially important to study the correlations between familial incidence and parental consanguinity, parents' ages and order of birth of children, and the clinical findings. Penrose[37] discusses the utilization of such data to contribute to the identification of rare mendelian recessive and rare mendelian dominant characters. He indicates that partially dominant genes which can cause mental or physical abnormality in parents are sometimes cumulative in their effects on children. Eugenically speaking, these dominants and recessives are vitally important, because it is likely that many more of them occur within the human race than are now known. Some strains may eventually prove to be responsive to biochemical therapy. Furthermore it may even become possible and advisable for families with such strains to prevent the production of offspring.

Many diseases known to medicine have a definite hereditary background. Keeler[25] has listed eighty-two of these diseases. Sachs[44], Davidenkov[6], Murphy[34], and others, have contributed descriptions of genetic deficiencies especially in relation to the fields of general medicine, neurology and mental deficiency. In all instances, however, we may think of genetic deficiencies as being responsible for establishing the organic basis for a large percentage of the mental or behavioral deficiencies of the human organism.

2. *Familial deficiencies.* — In this group belong those inherited deficiencies which are found manifested in family groups and in which no pathognomonic constitutional features have been determined. The prominent clinical features of the familial group are seen in general physical, mental and social inadequacies of the group as-a-whole. Specific organic deficiencies which may appear within the group may be a manifestation of the generalized deficiency of the organism, or of such specific strains of defect as may appear within any human group. The problem of familial deficiencies is of great social significance since such deficiencies represent a large proportion of the defectives produced by the high grade, borderline and subcultural groups. It has been customary in the field of mental deficiency to classify deficiencies on the basis of etiology into primary and secondary. Primary deficiencies are those for which the etiology is genetic or of unknown origin; secondary deficiencies include all cases *known* to have resulted from toxic or traumatic conditions occurring *after* fertilization. Familial types of deficiencies constitute the greater portion of the primary group and are distinguished from the secondary types by the fact that the latter are not due exclusively to heredity but arise from some injury of the living organism.

Tredgold[49], summarizing the findings of several authorities as to the frequency of primary defectives, has indicated that estimates of incidence of primary deficiency run on the average, from 70% to 90%. Meyerson et al[33] have reported that heredity plays an important role in this group. Wildenskov[51] has stated that the hereditary taint of mental deficiency is demonstrated considerably more often in the mild cases than in the severe (98% against 74%). Penrose[38] has observed that hereditary factors operated in 91% of his cases and that in 71% environmental factors were present. These figures indicate the overlap of hereditary and environmental factors. Penrose[39] has shown that disease conditions associated with hereditary mental defect have been most clearly demonstrated in idiots and imbeciles and that much less is known about hereditary factors in higher grades. In all grades of defect, however, heredity plays a part, but the degree of dominance of the hereditary factors may be different in various grades. He also expresses the opinion that defects in idiots and imbeciles are often recessively determined or are due to fresh mutation, and are less obviously hereditary than is the case with simpletons (higher grades), who like normal persons, owe their mental grade to the

interaction of dominant additive factors.

3. *Factors of Deficiency Related to Mental Illness.* — Recent studies of the genetics of mental illnesses extend the field of human deficiency well into the problems of mental illness. Duncan, Penrose and Turnbull[9,10], have called attention to the frequency of mental subnormality in the manic-depressive illnesses, the similarity of symptoms, the course and prognosis of these disorders in normal and subnormal groups, and the mood instability shown by many defectives, all suggesting a close association between the two conditions. They state that in certain cases, a manic-depressive psychosis may be "a manifestation of mental defect," and add: "We cannot frame our definition of the subnormal mind entirely within the limits of intellectual capacity, nor even within the impairment of such capacity as an essential feature. The defect in the manic may represent an impairment in the generating mechanisms of emotion, even in the terms of over-production of emotion and lack of governing factors. At least in the manic-depressive the defect lies primarily in the instability of mood." Slater[47] has questioned this relationship but nevertheless posits a specific dominant factor in the manic-depressive states. Rosanoff[43] has indicated that the bulk of manic-depressive cases have an hereditary basis.

Much evidence that schizophernia is frequently associated with hereditary factors is shown by Kallman[24] who states that "the probable incidence of schizophrenia among the offspring of schizophrenics is nineteen times greater than in the general population. Even the grandchildren, nephews and nieces are, in their turn, about five times as likely as the normal average person to become schizoid." Although Kallman[24] states that "Neither psychopathy nor congenital feeblemindedness have direct hereditary relations to the heredity-circle of schizophrenia, but spring from entirely different sources," the fact of basic underlying genic deficiency is again mentioned in relation to different types of mental deviations. James[17] supports Tredgold's contention that "primary mental deficiency, primary psychosis and dementia" are closely related and are "merely different manifestations of one and the same condition, namely, peculiarity of the germ material." James thinks this relationship is especially true in reference to primary mental deficiency and dementia praecox. Pollock, Malzberg and Fuller[41] also agree that hereditary factors play a definite role in the causation of both manic-depressive and schizophrenic behavior. The problems of psychopathological behavior in their relation to genetic causation lead to a consideration of Meyer's[32] psycho-biological concept of the organism functioning as a unit. This concept has contributed to studies of organic inferiority and of organ imbalance made by the students of constitutional medicine[36]. These and Lewis' morphological studies of dementia praecox patients[29], amply illustrate the importance of approaching the living organism as a functioning whole which is definitely influenced by predeterminant factors.

Since studies indicate a relationship of genetic deficiencies with mental illness, the importance of the genetic aspect of psychiatry becomes greater as the incidence of mental illness increases. The high incidence of these conditions is given by Landis and Page[26], who state that in New York "One person out of every twenty will at some time in his life become a patient in a mental hospital, while in all probability one out of ten will be incapacitated, though not sent to a state hospital, by mental disease, some time during his life." However, Dayton[7] observed in Massachusetts that recent statistics indicate only a slight increase in mental disorders — so slight as to allay "all apprehension."

These studies on the genetic factors in mental illness suggest that in some instances, mental illness may represent an expression either of primary or secondary mental deficiency. Mental deficiency, viewed from this interpretation, may be considered to constitute more than a type of intellectual deficiency, and may be more accurately termed "human deficiency." Any arrest in the processes of human de-

velopment, whether resulting from organic or from social factors, mav, in this sense, be described as a "developmental deficiency." Defective heredity, therefore, which is one form of human deficiency, furnishes a biological basis for more intimately relating the field of mental deficiency to many aspects of the medical and social sciences. The eugenical significance of such interrelationships is obvious.

B. Experimental Genetics

Another approach to the problem of genetics in relation to human deficiency is made through experimental procedures within the disciplines of physiology and psychology. In clinical or medical genetics, genic alterations exist as a result of the action of natural forces. In experimental genetics, genic alterations may be produced by a deliberate interference in genic relationships. This approach offers controlled conditions under which artificial dysgenesis, or normal or novel genetic reactions may be stutdied. The nature of experimental genetics is determined by the techniques involved, the material used, and by the training of the investigators.

1. *Physiological Approaches.*—Techniques in this subdivision of experimental genetics involve the deliberate use of chemicals and x-rays and have been employed largelv by biologists. From such work with the genetics of plant and animal life, much impetus has been given to investigators to extend genetical research into problems of the development of human beings. The need for correlated research between experimental genetics of this sort and medical genetics is especially emphasized when we call to mind the problem of so-called "bisexuality" or of "sex-intergrades" — a concept which bases on Goldschmidt's studies of sex-balance genes in moth hybrids. The works of Goldschmidt and Bridges (comment by Allen[1]), and of Lebedeff[28] on the genetics of certain invertebrates, the clinical work of Farrel[11] on human hermaphrodites, give color to Lang's theory[27] that homosexuality may be primarily a special case of intersexuality having a genetic basis. Such challenging leads are indicative of the importance of correlating basic genetic research with the clinical study of human psychosexual development.

2. *Phychological Approaches—With Infra-Human Subjects.* — Most of this work has been done by psychologists who have introduced unique techniques.

James[18] has already indicated that certain mammals — e.g., dogs, mav apparently inherit different organizations of their central nervous systems; that is, there may exist an intra-species variation in morphological and psycho-physiological reactivitv to stimuli — "Extreme aggressivity and shyness in social situations is correlated with excitability and inhibitibility as inherited constitutional factors." Both Heron[14] and Tryon[50], studying rats, have reported certain differences in the maze-learning ability of these mammals attributable to genetic factors. McDougall's "Fourth Report on a Lamarckian Experiment"[31] indicates the possibility of genetic improvement in strains of rats undergoing identical environmental experiences in successive generations.

b. *With Human Subjects.* — In spite of the importance of experimental genetics in physiological and animal-study approaches to the investigation of human development, the organism of man differs in many respects from the infra-humans. Many of man's particular types of reactions and needs must therefore be studied directly. This type of experimental genetics is also employed largely by the psychologists who use techniques not applicable to the research studies previously described. Such techniques include the use of standardized *psychological tests* on humans, and the use of specific *situational human experiences.*

The use of test-techniques in the study of genic factors of human development may be illustrated by the Rorschach test which bids fair to contribute greatly as a psychiatric-psychological tool in the study of psychical difference in individuals and in races. M. Bleuler[3] found remarkably similar responses to various Rorschach items from fraternal sibs and twins which had been reared apart.

These results together with other studies on identical twins, give strong indication that fairly specific mental traits, as well as physical ones, may be inherited. The Rorschach test has also been used by M. and R. Bleuler[4], and Hallowell et al[13] to study race differences. Doll's Social Maturity Scale[8] represents what may be the beginning of an important technique in the study of the inheritance of social competence. Other examples of the use ot test-techniques in relation to factors of heredity have been reported in *Mental Tests and Heredity* by Schieffelin and Schwesinger[45], and extension of this work may be found reported in Schwesinger's study[46] of the genesis of psychological characteristics.

The use of special situational experiences in order to study genetical implications of human behavior may be illustrated in studies by T. L. McCulloch[30] which undertake to link the results of certain work in primate biology with certain characteristics which are being investigated in the feebleminded. Attention has been focused upon those characteristics of human organization which presumably have been inherited from earlier stages of primate existence and which continue to influence present-day human behavior. Important forms of adaptation in defectives have been found which compare strictly with those of infra-human primates in similar situations; compared wth the great apes, certain patterns of adaptation of the lowest grade defectives are more primitive, while those of the middle grades are superior. Future work is directed towards characterizing the different types of adaptive behavior exhibited in standardized experimental situations by the variety of clinical subjects found at an institution, to the end that the patterns of adaptation may be analyzed in reference both to genetic implications and to social adjustments.

The studies cited in the preceding pages in no wise exhaust the important work that is being done. They are, however, representative of current investigations. These studies indicate the contributions of clinical and experimental genetics. The latter, especially, affords opportunity to explore patterns of mammalian behavior which may throw light upon human behavior and upon human deficiencies. In addition, direct observations of patterns of human behavior which may have a genetic basis promise much for the eugenics of the future.

C. Social Genetics

Studies in the genetics of society as-a-whole may not yet be organized as fully as are the fields of clinical or experimental genetics. One may think the use of the term "social genetics" as somewhat premature but a glance at certain steps now being taken in the study of population developments in our nation will give some warrant for the use of such a term. In fact, clinical and experimental genetics in themselves would be largely of academic interest were it not for their relationship to human factors in the phenomena of social organization. A defective Robinson Crusoe on a desert-isle could hardly represent the social challenge of the defective in the crowded isle of Manhattan. May we not say, then, that eugenics may be thought of as the field of "social genetics" wherein masses of human beings serve as subjects, wherein population studies furnish the data on behavior, wherein experiments in social control provide testing experience for eugenical control, and wherein the total field envisons the eugenics of the future in the terms of an international responsibility.

Spengler[48] and others have indicated that today's demographic selection is sociologically and apparently biologically dysgenic. The Population Index[42] is filled with numerous references to problems of human development in the terms of group activities.

It is encouraging to read of the attempts of various states to handle the problem of the defective in the terms of social genetics and governmental responsibility. Wilhite's report[52] represents one such attempt. Within our own State of New York, efforts are being made to develop a three-fold program in the interests of the subnormal groups which constitute so extensive a social and eco-

nomic problem. The program[51] includes the establishment of: a multiple-unit research unit wherein all medical and social sciences may work side by side over common problems; a joint state and university educational service — which will not only study defectives but will investigate problems of social organization among staff members, students, professional workers and the public-at-large; and, an experimental program of social control along the lines of preliminary studies now being made to determine what New York State might do to establish more effective control over its mentally defective and subcultural groups.

Comment

During the earlier days of social effort to provide care for mental deviants, these deviants were roughly divided into the categories of the feebleminded and the insane. Treatment procedures then, as of today, required the establishment of different types of institutions. However, scientific study of mental deviants has indicated closer interrelationships in the terms of inherited deficiencies. In special reference to mental deficiency, various investigators[16] use the term "mental deficiency" in relation, not only to cognitive deficiencies, but also to deficiencies of conation, affect-emotion, temperament, character and behavior. The enormous range in human deficiencies may be represented socially by: the "definitely ('true') feebleminded"—that is, the "low and middle grade defective" or the "idiots and imbeciles"; the "intermediate", "high grade", "border-line", "dull normal" or "subcultural groups"; the "psychopathic states" including the "moral defective"; "psychopathological states of deficiency" as found in the mentally ill; and deficiencies of special sense, or in mental association factors as found in every-day life. In the sense of developmental deficiency, wherein physical, intellectual, emotional and social deficiencies may be included, mental deficiency truly becomes a social problem of unmeasured magnitude. It may be said that all categories represent either temporary or permanent arrests in the growth of the total individual including both the organic and the psychological factors. The "true" mental defectives represent one general group of the developmentally arrested — that is — those who have failed to grow mentally or physically "from birth or an early age", and whose arrest is largely permanent.

No one person or single group can assume the responsibility for the treatment or elimination of the subnormal. As indicated by the 1940 Philadelphia Conference on the Registration and Social Control of the Mentally Defective and Subcultural[50], the interests of many of the medical and social sciences are jointly involved. The fields represented in the *American Journal of Mental Deficiency*[2] include genetics, biochemistry, physiology, physical anthropology, neurology, psychology, psychiatry, education and other social disciplines. The problems of human deficiency require that the field of eugenics become an integrating force among the scientific, the social, and the legal aspects of subnormality in reference to the general population.

The problem of human growth and development extends throughout all of human society and the study of clinical, experimental and social genetics affords opportunities for studying the deficiencies which may appear on a hereditary basis, in many different forms. "Mental deficiency" therefore means infinitely more than intellectual inferiority and the specific deficiencies in drive, emotion, character, personality and behavior of organisms within their various societies constitute by far the most serious aspect of problems in the field of human deficiency.

As Osborn[35] has stated in his excellent *Preface to Eugenics* — "The eugenics ideal recognizes that each human being is by his heredity unique. This uniqueness, which pervades every cell in his body, justifies respect for the individual. We cannot tell the heights to which any man may rise, until he meets the particular opportunity appropriate to his unique possibilities." Osborn clearly indicates that eugenics has become a problem of human government.

We may finally observe that life, which is an incessant striving toward levels of higher social integration is thus a challenge to eugenic ideals — from which we must not falter lest we, too, as individuals or as a group in a democratic society, become temporarily or permanently defective.

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ARE WOMEN PEOPLE?

Or Let's Not Cry Over the Random Assortment of the Chromosomes

REVIEWER'S NOTE: *This treatise started to be a book review. It ended up as something else—a listing and amplification of the reactions engendered by the book. The "reviewer" is sorry things got out of hand, and he hopes that the author, the publisher and the reader will not be too hard on him.*

LEWIS CARROLL'S Adventures of Alice have latterly taken an important place as texts in the newer physics and the newer semantics. Carroll's bedtiming was not all fun; he did a good job of dramatizing the limitations of our sense perceptions, and in calling attention to the dangers of jumping to merely verbal conclusions. In the field of social semantics, another unappreciated masterpiece has too long gone unrecognized: Hans Christian Andersen's fable of the Chinese emperor who wanted a new suit of clothes. The weavers spent a year working on a fabric so tenuous that neither the emperor nor any of his court were able to see it at all. Nevertheless when the suit was finally "fitted" the emperor set forth to show this much publicized sartorial masterpiece to his admiring subjects. It took a child of six to point out to his shocked mother that the emperor had nothing whatever on.

Andersen does not tell what happened to the boy. Doubtless he was soundly thrashed when his embarrassed mother got him home. And so he should have been, for he had brashly undertaken to call in question a set of concepts and conventions which a whole city had adopted and accepted. The point of this story today is that in the present critical times, our emergency is so grave that we must follow the lead of the skeptical infant and ask direct and embarrassing questions of the imperial humpty-dumpties who lead us in our processions and guide us in war or to peace, if there is to be a peace which will be more than a breathing space for bigger and better wars. To see that Emperor Humpty-Dumpty has nothing on, one must come either from outside the city or one must be too young and unsophisticated to accept the ponderous nonsense which sees the fruit of the loom when an invisible shuttle carried no warp whatever.

That the world is out of joint hardly needs to be labored. What to do about it is by no means a simple matter. The perplexing array of "cures" suggested only shows how unsure we are as to the basic causes of this surpassing and perplexing array of diseases which afflict us. A correct diagnosis must always precede a cure, and we must do better than merely to hope for a miracle as we try one alleged panacea after another. Good intentions have long ceased to be a justification for programs of social reform and there is hope in the fact that today they are hardly even an acceptable one-way ticket to Heaven.

In elaborating the complicated blueprint which is to guide us toward a better world, many diverse and conflicting areas of fact, belief, and convention must somehow be resolved. As a solvent of such conflicts, a eugenic point of view must become increasingly important. This biological approach conceives of man as a species "in being" which must conform to its environment without too much rattling around or too much friction. On account of man's unique social organization, his environment extends into a biological fourth dimension; the area of the intangible and imponderable mental, psychic and emotional aspects of culture. He can sicken and die of maladies of the spirit as well as of the body. Race suicide, that endemic disease of the vast majority of those who can and do read and write, is the premier eugenic symptom of this alarming sickness. To give us a realistic perspective of man in his environment, a eugenic point of view is essential in developing a rational understanding of how we have arrived

at the perplexing crisis we face as a species, and what may be done about it.

The Two Revolutions

This point of view will also help us to deal intelligently and humanely with the revolution that has engulfed us in the past few decades. This revolution is not a simple matter either. We have completely upset Man's physical environment through the ramifications of two surpassing revolutions. The Industrial Revolution has enormously changed the size and the shape of the world as far as our species is concerned. On top of that the Vital Revolution has completely altered man's patterns of living and dying over large areas of the earth's surface. Man has come to grips with his old enemy death so effectively in the last hundred years that how we are born, when we die, what we die of today differ vastly from the way our ancestors did these things. This complete upheaval in our fundamental biological pattern as a species must be studied and understood, and rationally and humanely controlled if we are to avoid disaster. The understanding and development of humane controls necessary to avoid the pitfalls likely to follow from such vast changes is no simple matter: In fact it is one of the most difficult ventures man has ever undertaken. To cast this most vital problem on the assumedly broad shoulders of "the blind forces of nature" or to saddle overworked Providence with it are neither of them evidences of intelligent behavior. If we are not willing to finish the job and come through to understanding and to humane and livable solutions, the future is hardly inviting.

The urgent importance of this aspect of the crisis is not even suspected by most people, including some of our leaders who are attempting to deal with the emergency. This challenge to our minds to use the machinery of science constructively for basic social understanding is the great challenge of our time. The defeat of totalitarianism and the problems of the peace which must follow it are only a facet of this greater problem, of really effectively coming to grips with life. This we must do because we have so successfully begun to come to grips with death in the Morning. (Death in the Afternoon is only secondarily a eugenic problem.)

". . . He Created Them"

One of the basic factors in this crisis is the perplexing question of marriage and the family and of the relations in 1941 society of men and women generally. And in this quarter of our world nothing much has happened during the machine-revolution or so far in the life-revolution. The rising sea of books on this subject testifies as to the bubbling and boiling in this quarter of man's environment, and to the need for some new and synthetic approaches. Recently added to the marital literary sound and fury is a book by Miss Pearl Buck which brings a novel outlook and which raises some interesting questions.* Miss Buck's upbringing in a missionary family in China gave her a unique opportunity to see two very different cultures at grips with the perennial man-woman question. Then after she had grown up in China she returned to America, the homeland of her parents, where she gained a wider outlook on Western marriage and sex mores.

This rare opportunity to become intimately acquainted with two very different cultures has resulted in a remarkable ferment in Miss Buck's thoughts and feelings. The reviewer is somewhat disappointed that a good beginning in developing a novel and stimulating point of view failed quite to come through to an integrated philosophy. She has over-simplified her human beings rather too much. Distressed that the differences between the sexes have too often been overemphasized, Miss Buck tends to swing too far the other way and to allege they differ merely in their anatomy and in no other respect. And in the process Miss Buck manages to throw several babes out with the bath. In recent years there have been great advances in the science of endocrinology which prove that the real differences between the sexes are

*Buck, Pearl S., *Of Men and Women.* Pp. 203. $2.50. John Day & Co., New York. 1941.

hormonal, and that the much publicized anatomical divergences are a weak reflection of these basic endocrinal difference which color our form and our feelings in diverse ways. That these differences have been cast in a stupid mold of "superior" and "inferior", of domination and acceptance, does not alter the fact that they exist, and that in blueprinting a new world they must be reckoned with.

Thus in her excitement about the world of men and women in America today Miss Buck has overlooked or ignored rather large areas. The reviewer suspects that she has compensated for this omission by verbally pawing the air to make up for this basic shortcoming. It smacks just a little of Andersen's naked emperor to pretend that men and women are duplicates, not complements, whose lives together should be at their best a theme with variations and overtones, not a sort of breathless double-talk. This gives us the possibility of a duet with harmony, rather than rather insipid exercises in the scale of C major on two pianos. The musician's art consists in the harmonizing of different tones, even of dissonances. The artist in life faces the same problem.

Some of Miss Buck's over-simplifications are a bit reminiscent of the disengenuous and disintegrating talk of "freedom" in the 1920's. But the reviewer is inclined to the paradoxical suspection that on the whole it will be better received by men than by women. As a somewhat perplexed male himself, the reviewer can find little that upsets him in the least. Perhaps this is because he had the curious experience of just having read what is perhaps a perfect introduction to this book. Susan Glaspell's *The Verge*. He understood that this was a fantasy about plant breeding, but stripped of a bizarre symbolism it is essentially a plea somehow to get through to reality in human relations. The "edge plant" which dared not push on into new fields of form and fragrance may be botanical nonsense but it is sound sociology. This brilliant and acrid portrayal of the conventional and traditional rejection of newness or of change throws into sharp relief Miss Buck's urgent critique. All of us too much resemble the tradition-bound sister who has so long been "a thief and a liar and a whore with words."

Bound Feet and Bound Minds

Miss Buck's observations really extend over three culture-patterns, because she has seen in China a complete revolution in the relations of the sexes. Little more than a generation ago women in China were puppets of the family, physically bound to the home by the wrappings which crushed their feet as babies. In spite of this they had a tremendous influence. Today in China women march beside men in the armies which face the Japanese invaders. As a result of this complete reversal in their position in society, Chinese women, with less opportunity than their American sisters, have nonetheless much more effective liberty than in this country. In the new capital of China there is a woman bank president; in America there are thousands of female bank clerks but no presidents of the fair sex: they rarely attain even the thirteenth vice-presidency. Which leads us to the strange paradox that today women are much more tradition-bound in this land of the free than they are in that ancient cradle of tradition across the Pacific.

Through their long history Miss Buck holds that the Chinese have attempted to deal factually and realistically and humorously with the world about them and with the people with whom they must live as relatives and neighbors. Since women have always been more numerous than men, and since marriage was conceded by the Chinese to be an honorable and desirable estate, polygamy was recognized not so much as a necessary evil as an inescapable inconvenience. Since women were destined to center their lives in the home, their training and their physical development were guided to this end. The footbinding was definitely a symbolical rite to typify the realities of the situation. In other ways the attempt was made to accept the facts of life and to deal with them as intelligently and humanely as possible. Mostly

the effort failed because it is impossible to have a people half slave and half free, which was the essential result of this system. Enslaved women as mothers of free man offered a continuing incongruity that wracked the system dangerously and engendered endless stresses and strains through the centuries.

In contrast with this situation in China, Miss Buck sees the American approach to the problem as essentially romantic and illusionary. The Chinese tried to be realistic, and out of that realism they produced a home life with many fine features and many consolations. Americans have gone at things quite differently. We have never clearly defined to ourselves what the real place of women in our society is, and we have trained them for a bogus adulthood which few of them—and they the exceptionally gifted—could ever realize. American girls have much greater freedom in their upbringing than Chinese girls. These beginnings would lead us—and them—to believe that they were to have a life of untrammeled freedom and unlimited opportunity. In this conventional picture there is only one traditional profession at which women are expected to excel: home-making (ignoring the mis-called oldest profession). She is sure that no serious attempt is made to train women for either of these vocations in our schools and colleges. And in her documentation of this view, Miss Buck cites the fact that in America nobody, and especially the women, expects a woman to be a leader in business, profession, or the arts. The predicament which this foggy fantasizing has brought us to is excellently symbolized in the works of Mr. James Thurber whose drawings of perplexed and frustrated people have become classics, and whose recent stage success, "The Male Animal," is nothing if not an epitomy of perplexed and futile rages—excellent comedy as an evening's entertainment, but grim in the extreme in proportion as it holds up the mirror to society.

Dissolving the Phantasy

All of this would be funny if it were not so tragic and pathetic, and if in this situation were not to be found some of the seeds of our present crises and perplexities. Out of this welter of traditionalized misunderstandings, Miss Buck sees both sexes groping in a fantastic blind man's bluff, varied by periods of increasingly urgent tension, relieved by the ultimate disaster of war. In a complicated world, people must somehow be able to return to simple unifying experiences—to contact with the basic realities of life and death. Childbirth, Miss Buck argues, furnishes this experience to women, and men on account of their physiological ineptness in this direction are driven to seek simplified contacts with reality in the madness of war. America's "gun-powder women" (as the author designates our growing army of spiritually and emotionally ill-clad, ill-housed, and ill-fed femininity)—driven to an ill-defined but nonetheless urgent desperation by stupid and irritational patterns which cast too many into a reluctant role as economic and intellectual zombies—have quite enough energy either to blow our entire culture into kingdom come or to rescue us from the brink of disaster. But to effect the miracle of this rescue, we must harness our eyes and our hearts and minds to building a more intelligent pattern of existence. Faser's *Golden Bough* bulges with the fantastic folklore by which men and women have contrived to misunderstand each other through the ages. Woman, Miss Buck points out, is no angel, and her bogus translation to this rank in the folk-lore of chivalry has hurt more than it has helped. Neither is she a devil, Mohammed and Saint Paul to the contrary, and the same goes for man as well.

Hardly anybody looks upon making a real home as an occupation in itself, a deplorable fact which the Census Bureau tacitly admits in its list of over six hundred occupations which leaves "housewife" or homemaker tacitly among the great unoccupied. In those halycon pre-gadget days when a man could work a half-dozen wives into early graves (in decent sequence, of course) the home was a much more robust institution than it is today. Miss Buck does

not feel this is all the woman's fault, and in that she is quite right. It is a symptom of the genius of our age, somehow to rob human life of meaning and social significance. The starving people of the Middle Ages sprinkled Europe with cathedrals which are an affirmation and a challenge. Our stupendous movie-houses, with a new super-collossal feature every week are a silly pipsqueak of response. The harnessing and expression of fundamental urges must somehow be re-effectuated. When that is done men and women will find it easier to get along, and to build meaningful lives. And when democracy rounds this corner, the Nazi menace, and the other corrosive menaces to human liberty will cease to terrify.

And the matter is not one merely of academic importance or of speeding the individual pursuit of happiness. In Germany, Miss Buck points out that the "gun-powder" women were among those who led the retreat to Nazism. In spite of a clear warning that "Kinder-Küchen-Kirche" meant a return to serfdom, all too many educated German women embraced these chains. She sees a real danger of a similar gruesome bellwethering in this perplexed land of ours. And if eugenics is to serve as the rallying ground for a modern crusade to capture a new heaven and a new earth, then the gun-powder girls must be enrolled, if such alarming trends are to be corrected. From more than one angle the need for the kind of revolution Miss Buck envisages is becoming evident.

One of the most encouraging and useful concepts of the newer eugenics is that in all races and in all social classes there is a huge diversity of genetic potentialities and disabilities, a diversity which far exceeds that found in any social or economic class. No group has a monopoly of all the hereditary virtues or of the vices and defects. The same thing can be truthfully and clarifyingly said of the sexes. The diversity of all kinds of characteristics is as great in one sex as in the other. To try to classify human beings with respect to qualities and abilities because they have two *X*-chromosomes instead of one is no more sensible than to claim that all the long headed people are among the chosen and all the round headed people are genetically and environmentally outcasts and pariahs.

Just at this point there is a place for a whole roomful of inspired social draftsmen who have the vision and the will to go courageously to work on problems of human diversity, which we have never really been done right by. God made man in his image, Scripture tells us, but *which man*? With becoming modesty we never have any real doubts about this: each of us is generously willing to take the job of stand-in, if not in person, our class surely. Photography, advertising, the schools, the movies and the radio have contributed their quota to an absurd process of standardization, with the "Arrow Collar Man" and "Miss America" as the ideals toward which each breathing blob of anthropomorphic clay is to be molded. The bracketing of differences as "superior" and "inferior" by some absurd yardstick is cruel and stupid, for there is abundant opportunity in a specialized society for a great variety of types and abilities. In fact, the standardization of human beings were it possible, would be a tragic disaster. It is high time once for all to strap old Procrustes to his own slumber-nook and leave him there. Then let us make up our minds to accept eagerly, to prize, enjoy and utilize that great wealth of varied talents and abilities which we find in our species as sex differences and as individual differences. The magic of the hormones makes of sex a mechanism for achieving greater diversity: this we cannot change by pretending otherwise. But we can change perfectly stupid conventions that some of these differences, whether they be conditioned by genes in *X*- and *Y*-chromosomes or elsewhere in the body genic, are *per se* to be looked down upon. Instead of making of this diversity a social hair-shirt, a tragedy and a ghastly waste, let us accept it and utilize it to the glory of mankind.—Robert Cook.

INBRED MICE

FOR RESEARCH IN EXPERIMENTAL BIOLOGY AND MEDICINE

DURING the past few years this Laboratory has completed the establishment of a number of inbred and hence genetically homogeneous strains of mice which have proven extremely valuable for research in experimental medicine and biology. The inbreeding is carried on through repeated brother times sister matings. The essential consequence of the inbreeding is that the individuals within a strain become increasingly uniform in their heredity and after twenty generations may be regarded for all practical purposes as genetically identical. The use of such material largely eliminates a common source of difficulty in biological investigations, namely, the inherent variability of the organisms ordinarily available. The uniformity of inbred strains is illustrated by the fact that tumors originating in such a strain, when transplanted, will grow in 100% of the individuals of that strain, but will grow irregularly if at all in other strains.

Careful checks are maintained on the homogeneity of the strains carried by this Laboratory. Sublines tend to develop within an inbred strain due to the accumulation of minor mutations. These are detected and where advisable eliminated through the use of appropriate tests and breeding methods.

The Laboratory will welcome inquiries from scientific laboratories and hospitals concerning the inbred stock available or with regard to the possibility of providing stocks and types suitable for special experimental problems.

Supply Department

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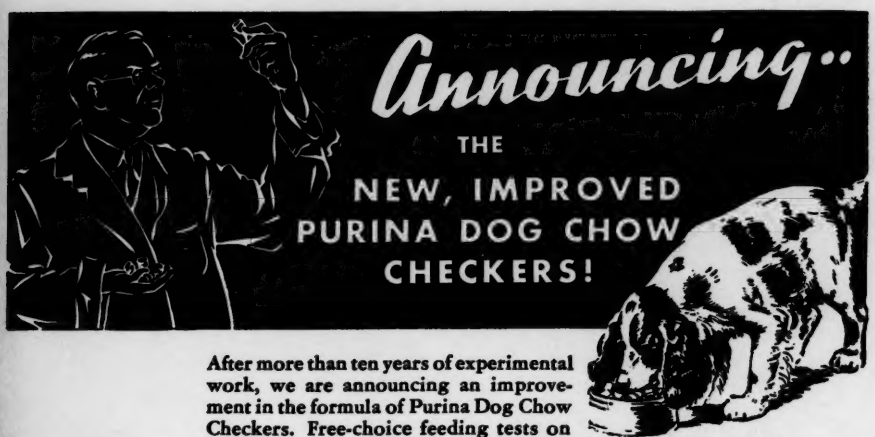
Bar Harbor **Maine**



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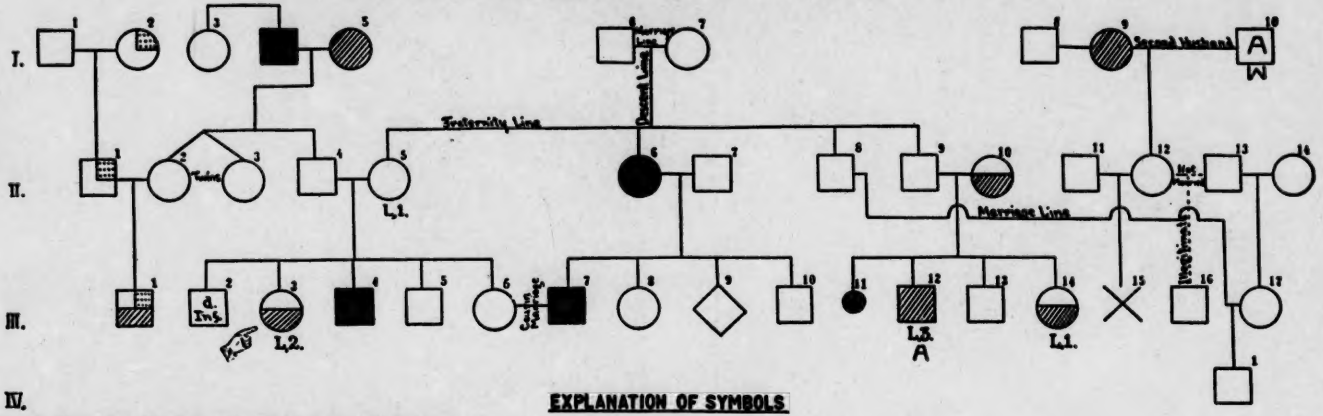
SAMPLE PEDIGREE CHART SHOWING THE MANNER OF CONSTRUCTION, AND THE USE OF STANDARD AND SPECIAL SYMBOLS.



EXPLANATION OF SYMBOLS

(a) **Standard for all Pedigree Charts:**

□=Male; ○=Female; ◇=Sex unknown; ●=Still-birth or Miscarriage; X=Children — number and sex unknown; ○○=Twins; d. Inf.=Died in Infancy; Points to the Propositus or central figure in the pedigree.

□—○=Parents.

□○□○○=Children.

Roman figures to the left indicate generations; Arabic figures locate individuals, (thus III. 7 is the young man in the third generation who married his cousin).

The following letters, placed in or around the individual's pedigree symbol, are standard for certain traits: A, alcoholic; B, blind; D, deaf; E, epileptic; F, feebleminded; I, insane; M, migraineous; N, normal in reference to traits under consideration; Ne, neurotic; P, paralytic; Sx, sexually immoral; S, syphilitic; T, tuberculous; W, wanderer.

(b) **Especially devised or selected for this particular pedigree-chart:**

■ ● = Successful leaders in politics. ◫ ◷ = Extra thumb on right hand. L.1. = Highly successful author. L.3. = Little or no ability in literary efforts. ▨ ◍ = Superior in vocal music. ▨ ◍ = Medium attainment in vocal music.

To fit the particular family and traits (whether physical, mental or temperamental; good or bad) under consideration, invent special symbols, or select special letters (in addition to those standardized under (a) above) to be placed within or near the particular individual's pedigree symbol, to indicate particular traits and their degree of development.

Prepared by the International Commission of Eugenics. See *Journal of Heredity* for August 1926. Additional copies may be obtained from A.G.A.